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CONTENTS OF PREVIOUS NUMBERS

SEPTEMBER, 1923. NUMBER 3

OCTOBER, 1923. NUMBER 4

Archives of Neurology and Psychiatry

Vol. 10 NOVEMBER, 1923 No. 5

CENTRAL PAIN IN SYRINGOMYELIA AND DYSESTHESIA AND OVERREACTION TO SENSORY STIMULI IN LESIONS BELOW THE OPTIC THALAMUS *

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Pain has been frequently observed in syringomyelia and has been attributed to implication of posterior roots, as by meningitis. This explanation is satisfactory for some cases but not for all, and some other explanation for some of these cases must be found.

Oppenheim [1] has not much to say about pain in syringomyelia. He remarks that pain is not uncommon and that French writers (Raymond and Lhermitte) describe a special form of syringomyelia characterized by intense pain. The cause of this pain he does not mention.

H. Schlesinger [2] mentions that pain may be intense, so great as to take away all joy of life. He has observed severe pain especially in cases in which later disturbances in the motor and trophic functions developed. The pain may be boring, tearing, may simulate chronic rheumatism, or may be lancinating as in tabes. In one of his cases with pain, tabes also existed; in another a large tumor; in another the symptoms indicated meningitis; and Westphal, Junior, found leptomeningitis associated with syringomyelia. These conditions are so mentioned as to imply that Schlesinger sought in them the cause of the pain. Irritative sensory symptoms he has found more common in the upper than in the lower limbs, because of the prevalence of the lesion in the cervical and upper thoracic region, and they are more frequently unilateral.

Haenel [3] speaks of pain in syringomyelia and also considers it more commonly unilateral. When root changes are wanting he believes

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* Read before the Forty-Ninth Annual Meeting of the American Neurological Association, Boston, May 31 to June 2, 1923.

* From the Philadelphia General Hospital and the Neurological Laboratory of the University of Pennsylvania.

1. Oppenheim, H.: Lehrbuch der Nervenkrankheiten, Berlin: S. Karger, Ed. 5, p. 435.

2. Schlesinger, H.: Die Syringomyelie., Vienna: Ed. 2, 1902, p. 27.

3. Haenel: Lewandowsky's Handbuch der Neurologie **2**: part 1, 587, 1911.

these pains show that the intramedullary pain tracts may be excited, but when pain is of long duration a complicating meningitis is to be diagnosed.

Dejerine and Thomas [4] speak of syringomyelic pains, regard them especially as an early sign, but do not explain them.

A case of intense pain in the lower limbs in syringomyelia has recently been under my care, and microscopic examination has shown that the pain could not be attributed to meningitis or irritation of the posterior roots.

REPORT OF CASE

Clinical History.—R. S., a colored woman, aged 60 years, entered my service in the Philadelphia General Hospital, Dec. 23, 1922, and died Jan. 19, 1923. She complained of weakness of the right upper and lower limbs and of less weakness of the limbs of the left side. The condition had been developing gradually for six years, and she had been unable to work in the last two or three years. She frequently burned her hands and did not know she had done so until she saw the blisters. The weakness had developed first in the right upper and lower limbs, and she dragged the right foot; the weakness developed so gradually that she was able to get about with a cane until about a week before she entered the hospital. The left hand had been growing weak during the previous six months, as had also the left lower limb, though to a less degree.

She had been having much severe pain in the lower limbs during the previous year, and it began in both lower limbs at the same time. It had been especially severe for six months. The pain was dull and aching, constantly present and spontaneous, with the exception of remissions of days at a time. The intern on my service, Dr. Maeder, to whom I am much indebted for assistance in obtaining these notes, and the nurses in charge of the patient observed that the pain was worse when the patient was handled. This was evidently hypersensitiveness to pressure.

On December 23, 1922, she fell and since that time was unable to walk or move her lower limbs to any decided extent. The right lower limb became completely paralyzed and the right upper limb weaker. Blisters developed on the inside of each thigh, and appeared suddenly during the night. They were evidently trophic.

Neurologic Examination.—No abnormal condition was found in the head. The pupils and ocular movements were normal.

The left upper limb was weak, but was by no means paralyzed. It is recorded that no pronounced sensory disturbance was detected in this limb for touch, pain, heat or cold, or position sensations, but one must doubt whether some impairment of sensation did not exist. The interosseous and thenar muscles showed some atrophy.

The right upper limb was much paralyzed, but the extensor muscles on the forearm and the muscles of the upper arm were better preserved. The muscles of the shoulder girdle also were weak. The shoulder joint could not be moved. The tendon reflexes of this limb were lost. Pain sensation was markedly diminished in the right upper limb and over the scapula. Tactile

4. Dejerine and Thomas: Traité des maladies de la moelle épinière, 1902, p. 217.

sensation also was lessened in these parts. Position sense was preserved. The right hand showed considerable puffiness but no true edema. It suggested the "main succulente" of Marinesco.

The left lower limb showed no paralysis, atrophy or tremor, but gave pain on pressure. Babinski's sign was present. There was no clonus. The patellar reflex was active. Heat and cold sensations were impaired. The right lower

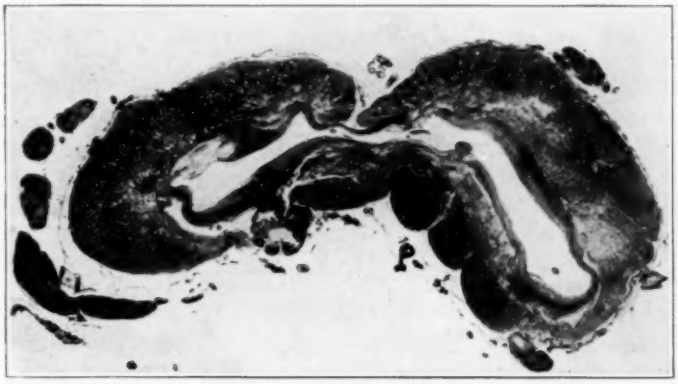

Fig. 1.—Section at the seventh cervical segment. A considerable part of the periphery of the cord containing the spinothalamic tract is intact. *P*, posterior septum.

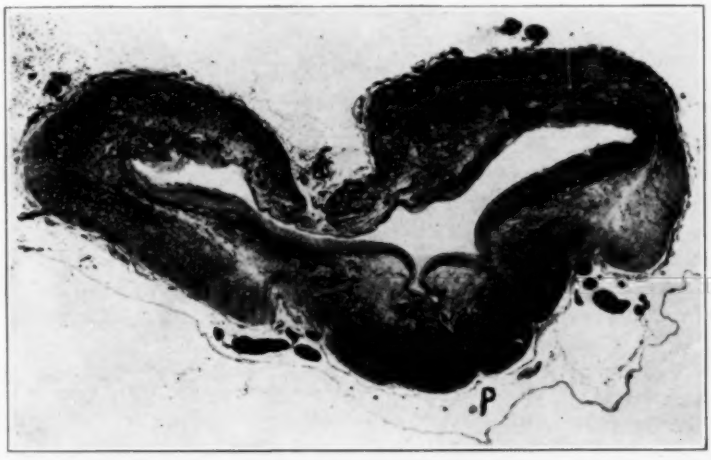

Fig. 2.—Section at the eighth cervical segment.

limb was completely paralyzed but not atrophied. Marked pain was obtained on pressure. Babinski's sign was present. There was no clonus. The patellar reflex was exaggerated. Heat and cold sensations were distinctly impaired.

The case appeared to be one of syringomyelia.

The woman died Jan. 19, 1923, from cardiac weakness, pulmonary edema and acute nephritis.

Microscopic Findings.—The microscopic examination gave the following results:

The cavity extended into the medulla oblongata but was confined to one side and to the course of the ninth and tenth nerves within the medulla oblongata, as is frequently seen when syringomyelia extends upward. It followed the course of these nerves into the spinal root of the fifth nerve. It did not extend high enough to cause serious degeneration of the median lemniscus.

The uppermost part of the cervical region was not obtained at the necropsy. The cavity was pronounced at the seventh cervical segment. The cord was much distorted, and the cavity with its dense glial wall implicated both posterior horns and the anterior part of the posterior columns. The anterior part of each anterior horn escaped. The anterolateral columns were partly degenerated. Both crossed pyramidal tracts were much degenerated. The nerve cells of each anterior horn were greatly degenerated, and those of the left horn more than those of the right horn.

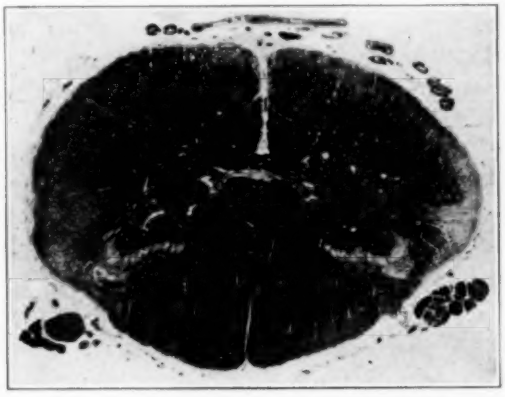

Fig. 3.—Section at the second lumbar segment. The spinal cord, except for secondary degeneration, is intact.

The condition at the eighth cervical segment was similar to that at the seventh cervical segment, but the degeneration of the right lateral and right anterolateral columns was more intense than of the left similar columns, and more intense than in the seventh cervical segment.

The findings at the first thoracic segment were similar but the cavity was somewhat smaller than in the segment above.

The midthoracic segments presented much the same appearance as the first thoracic segment.

No cavity was found in any of the lumbar segments. There was no meningitis anywhere in the cord. The lumbar sections showed only the secondary degeneration of the crossed pyramidal tracts. There was a very slight lymphocytic infiltration in the lumbar pia, but it was too insignificant to have caused symptoms. The posterior lumbar roots were not at all degenerated.

I have been interested for a long time in dysesthesia and overreaction to sensory stimuli from lesions below the medulla oblongata, and also

in pain produced by lesions below the thalamus. In 1908 I referred [5] to the occurrence of subjective sensory disturbance of sensation, including the presence of pain, in occlusion of the posterior inferior cerebellar artery. Some evidence on this matter is afforded in the literature.

Lewandowsky [6] believed that central pain in disease of the spinal cord in the majority of cases is caused by irritation of the posterior roots and that pain can be produced by any condition of the central tracts he says has long been doubted or denied. The pains of syringomyelia are to be attributed to irritation of the posterior roots. Hematomyelia may cause pain at its onset, which possibly is the result of irritation of the posterior roots. Pain usually is absent in intramedullary tumor, but not always; especially may it occur when the tumor by distortion of the spinal cord causes irritation of the posterior roots. Pain is rarely observed in multiple sclerosis; when it occurs, it is the result of irritation of the posterior roots within the spinal cord. The pain in all these conditions is local and segmental.

In rare cases he acknowledges pain is caused by irritation of the intramedullary tracts, as when an intramedullary tumor causes pain on the opposite side of the body, and it may exist with diminished objective pain sensation. He referred to Oppenheim's description of spasmodynia cruciata, i. e., pain on the side of the body opposite the lesion and tonic spasm on the same side as the lesion. Lewandowsky accepts pain of thalamic origin and refers in this connection to Greiff, Lauenstein, and Edinger.

It may be, he thinks, that so-called central pain is not always central, but may result from a change in the pain threshold for irritation originated at the periphery. Usually central pain is associated with hypesthesia; Lewandowsky has always found it so associated, and thereby a delay is produced in pain conduction. One so affected does not feel touch or pinprick of brief duration, but does when the contact is longer. Warmth may not be felt on the affected side when the contact is brief, but may be painful by longer contact when it is not painful on the unaffected side. Some patients experience pain when they lie on their hemiplegic side, and this is very similar to central pain. It is possible that central pain may result from abnormal summation in partial interruption of the sensory tracts, possibly from overstimulation of pain centers.

Pain is rare in tract disease, as in Friedreich's ataxia. The pain attacks which sometimes precede the onset of hemiplegia (Weir Mitchell, Féré) are to be attributed to irritation of pain fibers and cortical pain centers.

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5. Spiller, W. G.: The Symptom-Complex of Occlusion of the Posterior Inferior Cerebellar Artery: Two Cases with Necropsy, J. Nerv. & Ment. Dis. **35**:377, 1908.

6. Lewandowsky: Handbuch der Neurologie, **1**: Part 2, 800, 1911.

Gordon Holmes [7] has made a study of pain of spinal cord origin. He says that the histologic changes in the cord which produce this pain may not interrupt conduction in the white matter which they affect. That they may modify the impressions that pass through the affected fibers seems very probable, and it is to this he attributes the alterations in the sensations evoked by stimuli in the hypersensitive regions. The so-called spontaneous pains, in so far as they are independent of peripheral excitements, are probably due to the irritative effects of these lesions of the conducting fibers; and the rarity of these pains probably depends on the infrequency of that type and degree of pathologic change that can irritate the fibers and yet not block conduction through them. Holmes cannot explain why the peculiar lesions producing pain are so rare.

His explanation hardly fits in with the explanation he and Head gave for pains of optic thalamus origin. They believed that peripherally projected pains are not, as has been generally assumed, due to irritation of central conducting tracts, but are a consequence of the removal of the inhibitory control which the cerebral cortex normally exerts on subcortical centers that are concerned in the perception of this form of sensation. They assumed that this center lies in the inner division of the optic thalamus. One can hardly apply this theory to pains of spinal cord origin.

Up to the time that Holmes wrote his paper, he could say that practically the only other central lesion than that of the optic thalamus which produces similar pain is disease of the medulla oblongata and the lower part of the pons. He refers to Mann's case, in which the lesion was in the right side of the bulb, and says that he (Holmes) has had a man under his care in whom the disease of the same region caused similar pain.

Holmes' patients with war injuries, in whom he observed the pains of spinal cord origin, had spontaneous pains of intense severity which could not be attributed to lesions of posterior roots, but they could be excited and increased by all peripheral stimuli. Even the light contact of a finger or the touch of a wisp of cotton wool evoked or aggravated the pain, and patients dreaded particularly movements of the affected parts. Even a slight jar to the bed might bring on a bout of intense pain. Holmes had not seen, during more than four years' work in military hospitals, any other condition associated with such intense suffering. In his cases, the suffering was only temporary and the pain subsided toward the end of the second or third week. In sixteen

7. Holmes, Gordon: Contributions to Medical and Biological Research, Dedicated to Sir William Osler.

cases the pains were referred to distant regions of the body below the level of the wound. In a few cases the pains persisted during the six or seven weeks that the men remained under observation.

The case reported by Mann,[8] to which reference has been made, was purely clinical and the diagnosis was made from the clinical findings, it was encephalomalacia medullæ oblongatæ.

In another well-known case, a case of tubercle of the left side of the pons, reported by Economo,[9] severe pains had been present in the entire right side of the body and were relieved only temporarily by morphin. The pain was attributed to a lesion of the tractus spinothalamicus et tectalis, and Economo believed his case demonstrated that a lesion considerably below the optic thalamus could cause central pain. The case was with necropsy.

I have observed dysesthesia produced by an acute lesion of the spinal cord, such as myelitis, as a burning sensation from a touch or pinprick when either form of irritation was recognized with difficulty. It is similar to the causalgia in certain peripheral nerve injuries, but the form to which I am referring is not a spontaneous burning sensation but is produced by peripheral irritation. Why this burning pain is present in some cases and not in others apparently very similar, I do not know.

I have also observed hypersensitiveness to pressure in lesions of the central nervous system. It was present in the case of syringomyelia described in this paper, and was also present in a case in which the lesion evidently was in the pons, to which I shall refer presently. In the latter case the patient could not stand the pressure of the bedclothes on the affected limb. This increased transmission of sensation (over-reaction) in central lesions is interesting; pain on pressure in such a case does not establish neuritis, and cannot be explained as a complicating neuritis. The nerve cells of the anterior horns in my case of syringomyelia did not show degeneration as they probably would have done had neuritis of long duration been present.

A very remarkable example of over-reaction to peripheral stimuli in the face, a case of striking dysesthesia, was reported[10] by me in 1915 in a paper entitled "Remarks on the Central Representation of Sensation." The lesion was almost certainly below the optic thalamus. The important features of the case were:

> The sudden onset of analgesia in a syphilitic man, that was confined to the distribution of the left trigeminal nerve, without any other form of objective disturbance in this distribution; and the paresis of the left soft palate

8. Mann: Berl. klin. Wchnschr., 244, 1892.

9. Economo: Jahrb. f. Psychiat. u. Neurol. **32**:107, 1911.

10. Spiller: Remarks on the Central Representation of Sensation, J. Nerv. & Ment. Dis. **42**:406 (June) 1915.

and left vocal cord. There seemed to be scarcely any doubt that the lesion was a vascular one in the left side of the medulla oblongata, implicating the spinal root of the left trigeminus nerve and also the glossopharyngeus and vagus nerves. This man had had a sudden onset of paresthesia limited to the left fifth nerve distribution. He had been in a barbershop and after he left the shop the left side of the face felt numb. He attributed this at first to soap which he thought the barber had left on his face, and he attempted to remove the sensation by washing the face. The paresthesia was one of a sensation of stiffness, burning or numbness. It was not in the lower part of the face in the distribution of the cervical nerves. He could not permit a fly to be on the left side of his face, and he said the sensation produced by a fly was "a tickling all over the left side of the face." Tickling the left side of his face by light contact with the end of a cloth was very annoying to him. A fly was caught and made to crawl over the left side of his face, and the man showed intense dysesthesia and discomfort and distorted his face greatly. There was thus marked over-reaction to sensory stimuli from a lesion of the medulla oblongata.

I called attention to the fact in 1915 that this case did not support the statement of Head and Holmes that over-reaction to sensory stimuli can be attributed alone to disturbed activity of the optic thalamus. In this case there was at first some pain over the left eye which may have been central pain.

Another interesting case which showed over-reaction to sensory stimuli from a lesion presumably in the pons is the following:

J. L., aged 51 years, consulted me first, Aug. 29, 1911. He had been well until eighteen weeks previously when he developed a constant headache, worse on the right side, which began in the frontal region but soon extended to the top of the head. The most distressing feature was a series of sharp shooting pains in the right posterior auricular nerve distribution. Four weeks later the headache became much more intense, and vertigo and nausea occurred; within twenty-four hours these symptoms became so intense that he was unable to stand; and the following additional symptoms were noted: diplopia, hypesthesia to pain and temperature throughout the entire left half of the body most intense in the left lower extremity, numbness throughout the entire left side, and a marked incoordination of all four extremities—more intense on the right side. Diplopia persisted for only a week. The incoordination diminished after the first ten days, and the vertigo after two weeks.

Within three weeks after the onset of his symptoms he developed a burning pain in the left foot which gradually grew more intense and extended to the leg. After two more weeks, when he got out of bed, the burning pain in the left lower limb became very intense and an aching pain developed in the left upper limb. These pains had persisted ever since their onset, though they had become somewhat less severe. In spite of the left-sided hypesthesia, he had an intensely unpleasant emotional response to stimuli on the left side. He was annoyed by the pressure of the bedclothes on the left side, he could scarcely endure having the toe-nails of the left foot trimmed, and he was annoyed by the rubbing of his clothing on the left side of the scrotum. He had become peculiarly sensitive to noises. On his way East he was in agony when his train passed another train, and even at the time of examination the slamming of a door "Hurt him in the left side."

In addition to these sensory changes he had slight motor weakness on the left side, most marked in the lower extremity. He could walk only about 150 yards when he was obliged to stop, not because of the pain but because of weakness. He had no athetoid movements and had control of the vesical and rectal sphincters.

In my examination I found distinct hypesthesia throughout the sensory distribution of the right fifth nerve, sharply confined to this distribution. Unfortunately the physician recording this finding failed to mention in the note what form of sensation was tested. I can be certain only that he had disturbed objective sensation in the right fifth nerve distribution. Sense of taste was lost on the right side of the tongue. A slight but distinct paresis of the face of the central type was present on the left side. The right conjunctival and corneal reflexes were diminished. The tongue was protruded slightly to the left; the soft palate was moved normally on each side.

The left upper and lower limbs when examined were possibly a little weak. Hypesthesia for pain, heat and cold sensations were present throughout the left side, marked in the lower limb to about the knee, slight in the thigh, and very slight above that level. Tactile sensation was normal on the left side. Incoordination was distinct, and was more intense on the right side. The finger to nose test was performed by the left upper limb with almost normal coordination, but it was abnormal on the right side and showed slight hypometria. Stereognosis was good. Station with eyes open gave only a slight sway, but the man tended to fall when he closed his eyes. Gait was with short steps and feet rather wide apart.

On account of the sharply defined left hemihypesthesia, the constant left-sided pain, the unpleasant and exaggerated emotional reaction to left-sided stimuli (dysesthesia and over-reaction) and slight suggestion of transient left hemiparesis, the lesion was placed in the right side; and the implication of the sensory root of the right fifth nerve, shown by hypesthesia, the loss of taste on the right side of the tongue and the diminution of the right conjunctival and corneal reflexes, indicated the level of the lesion as in the right side of the pons or medulla oblongata. The motor fifth nerve was not implicated on either side, which would suggest that the lesion was not exactly at the level of the main division of the fifth nerve in the pons.

The blood pressure was unusually low, 100 systolic and 76 diastolic.

CENTRAL PAIN FROM LESIONS OF THE PONS*

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While pain and painful paresthesia have long been known as symptoms of thalamic disease, it has always been a question among neurologists whether these symptoms could occur from involvement of the central nervous system below the optic thalamus. The pain and uncomfortable paresthesia caused by disease of the thalamus, have been explained by most observers as being due to irritation of fibers of the sensory tracts, but Henry Head [1] believes that central pain is the result of setting the thalamic center, located in the medial nucleus, free from cortical control, with the result that it acts without restraint to all stimuli capable of arousing affective states. According to him the cortical sensory fibers end in the lateral nucleus, the region usually affected in cases exhibiting the thalamic syndrome.

Head states that he "has not found this over-response to stimuli, as well as spontaneous pain, from lesions of other parts of the sensory path," and calls attention to the extreme rarity of such cases in the literature compared with the marked frequency of this over-response in lesions of the optic thalamus. Of the reported cases of lesions below the optic thalamus causing central pain, Head thinks few only will stand criticism.

In 1922, in discussing central pain, Spiller [2] said: "I should like to have satisfactory evidence that a lesion entirely below the optic thalamus may cause spontaneous pain in one half of the body. Gordon Holmes has spoken of central pain from lesions of the spinal cord, but I am unable to refer to any reliable case in which a lesion entirely sub-thalamic had caused intense unilateral pain, affecting both upper and lower limbs."

We have long thought that central pain may occur from irritation of sensory fibers in any portion of their intramedullary course, and there are abundant cases in the literature to support this view. For

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* Read at the Forty-Ninth Annual Meeting of the American Neurological Association, Boston, May 31, June 2, 1923.

1. Head, Henry: Studies in Neurology, New York: Oxford University Press, Vol. 2, p. 597.

2. Spiller, W. G.: Arch. Neurol. & Psychiat. **8**:215 (Aug.) 1922.

example, Gordon Holmes [3] has shown that traumatic injury of the pain conducting paths, within the spinal cord, causes central pain. He described sixteen cases of gunshot wounds, in which pain was referred to distant regions of the body below the level of the wound, and stated that the rarity of central pain was probably due to the infrequency of the type and degree of pathologic change that can irritate the fibers, and yet not block conduction through them. Holmes further made the interesting observation that lesions of the dorsal columns can produce central pain, since vibration of the tuning fork, in some of his cases, was painful on the homolateral side.

Cases of intramedullary and extramedullary spinal cord tumors, with referred pain not due to irritation of the posterior roots, have been reported. In addition, cases of syringomyelia producing central pain, with necropsy, have been described by Taylor, Greenfield and Martin.[4]

Central pain from lesions of the medulla oblongata is common. While central pain has been reported in numerous cases with a clinical diagnosis of occlusion of the posterior inferior cerebellar artery, so far as we know, the only cases in which this was confirmed pathologically are those of Senator,[5] Wallenburg,[6] Thomas,[7] and Hun.[8]

Before discussing lesions of the pons, we shall refer briefly to thalamic, cortical and subcortical cases. Extended reference to the thalamic literature is hardly necessary, for neurologists are familiar with this symptom complex.

There are a few undoubted instances in the literature of lesions in the cortex and subcortex that caused central pain.

While Head does not altogether subscribe to this point of view, nevertheless from the following statement such an assumption can be made: "Cortical lesions produce no true raising of the threshold to prick, unless it is extensive or is associated with subcortical destruction. It may, however, induce a change in the sensation produced by pricking, which is recognized by the patient. Sometimes the whole character of the sensation is said to be changed and seems to tingle or to resemble electricity."

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3. Holmes, Gordon: Central Pain. Contributions to Medical and Biological Research, London: **1**:235, 1919.

4. Taylor, James; Greenfield, J. S., and Martin, J. P.: Two Cases of Syringomyelia and Syringobulbia Observed Clinically Over Many Years and Examined Pathologically, Brain **44**:323, (Parts 3 and 4) 1921.

5. Senator: Arch. f. Psychiat. **11**:713, 1881; and **14**:643, 1883.

6. Wallenburg: Arch. f. Psychiat. **34**:923, 1901; and **27**:504, 1895.

7. Thomas, H. M.: J. Nerv. & Ment. Dis. **34**:48, 1907.

8. Hun: New York M. J. **1**:513, 1897.

LESIONS IN THE PONS

Pontile lesions causing central pain are very rare. We have been able to find in the literature only two clinical and two pathologic reports. Of the clinical cases, in one reported by Raymond and Rose [9] as a lesion of the pons, the authors admit the possibility of multiple lesions; in the second case, reported by Raymond and Français,[10] pain and hyperesthesia to pinprick were present.

Of the two pathologic reports, one, by Economo,[11] is a case similar to ours; the other, by Mills, was a case of occlusion of the left superior cerebellar artery. The patient in the latter case had a sense of numbness or impaired sensation throughout the entire right half of the body. Careful tests showed that sensation of all forms was fully retained on the left half of the body, but was lost to pain, extreme heat and cold on the right half of the body. Tactile discrimination was also greatly impaired as was shown by the compass test. Light touch was preserved. The senses of deep pressure, position and passive movement were normal on both sides, and the patient showed no astereognosis in either hand. This case, studied pathologically by Spiller [12] showed, among other lesions, at the level of the mesencephalic root of the fifth nerve, degeneration of the lateral lemniscus and of a small part of the tegmentum of the pons, medial to the superior cerebellar peduncle. There was also degeneration of the superior cerebellar peduncle, with the exception of a small part near the medial lemniscus.

Because of the resemblance of Economo's case to ours, we cite his case at length.

The patient, a man, 35 years of age, first complained of inability to recognize heat in the right leg. Soon afterward he complained of paresthesia in the right upper and lower limbs, and buzzing in the left ear. Examination showed that the pupils and the ocular movements were normal. There was slight nystagmus, chiefly to the right, with no paralysis of associated ocular movements. The eyegrounds were normal. There was left motor fifth nerve palsy, with sensory disturbance in all branches of the fifth nerve, chiefly in the upper branch; later there developed a left seventh nerve palsy. The reflexes were normal, though possibly weaker on the right side. Motor power, with the exception noted, was never disturbed. Talking, eating and swallowing were always normal.

There was loss of pain and temperature sensibility on the entire right side. Later the patient had disturbance of taste in the left side of the tongue, as well as of heat sensibility in the right trigeminus, but touch sensibility was not impaired in this part.

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9. Raymond and Rose: Syndrome de la calotte protubérantielle, Rev. neurol. **16**:263, 1908.

10. Raymond and Français: Syndrome protubérantielle avec hyperexcitabilité du nerf facial et troubles du goût. Rev. neurol. **17**:445, 1909.

11. Economo, E.: Jahrb. f. Psychiat. u. Neurol. **32**:109, 1911.

12. Spiller, W. G.: J. Nerv. & Ment. Dis. **42**:412, 1915.

At no time did the patient have disturbance of tactile or deep sensibility. Astereognosis apparently was not present, as it is not mentioned. The patient complained of pain in the right side of the body, which was so severe that he could not sleep and had to be given morphin. The pain was not only present in the right arm and leg, but was so marked in the chest as to interfere with breathing, and was like colic in the abdomen.

Macroscopic and microscopic examinations demonstrated a tuberculous lesion, the lower limit of which was at the entrance of the left acoustic nerve. The largest part of the lesion was in the pons at the region of the sensory fifth nerve nucleus. The tumor displaced the lateral part of the pons only, its upper level being in the superior lateral part of the pons, not involving the anterior cerebellar peduncle or the lateral portion of the medial lemniscus. The tubercle destroyed the left eighth nerve and the greater portion of the vestibular tract of Deiter's nucleus; the motor and sensory portions of the trigeminal nerve were severely involved. The sixth nerve was medial to the tuberculoma, its nucleus being free. The other cranial nerves were normal. In addition, there were involved: a portion of the corpus restiforme; the greatest part of the substantia reticularis lateralis tegmenti and the fibers which go through it; the spinotectal and spinothalamic tract; and the rubrospinal bundle. The region between the sixth nerve and the raphe, which includes the medial lemniscus, the fibrae praedorsales, and the posterior longitudinal bundle, was intact.

We report here two cases, the first with clinical history only, the second with pathologic findings.

REPORT OF CLINICAL CASE

CASE 1.—*History.*—A physician, 42 years of age, after a severe headache followed by vertigo, experienced numbness in the left arm and leg, these limbs feeling as if they were "thick and asleep." At the same time the right side of the face felt "queer." Coincident with this, he complained of seeing double. There was also marked hyperacusis; all noises seemed to be intensified.

Neurologic Examination.—An examination made six weeks after the onset revealed paralysis of the muscles of mastication on the right side, the jaw deviating to the right on opening. There were no contractions of the temporal and masseter muscles on that side. The right sixth nerve was paralyzed and the right seventh nerve was paretic. There was no paralysis on the left side of the face, but slight weakness was present in the left arm and leg. The reflexes on this side were more prompt, this being aspecially true of the knee and Achilles jerks, and on plantar irritation the large toe had a tendency to move upward.

At first the patient did not recognize objects in the left hand, but this astereognosis was less marked on subsequent examinations. Sensory examination at no time showed total loss of the common forms of sensibility, but in the beginning there was considerable impairment of all forms of sensation on the left side. This can be accepted without question as the patient was an intelligent physician. When first examined by one of us, he had disturbance in sense of position and movement in the left upper and lower limbs, particularly the upper, and in the finger-to-nose and heel-to-knee tests there was distinct ataxia, especially in the upper limb. At this time there was disturbance

of tactile and deep sensibility in the left upper and lower limbs, and also of heat sensibility, but tests for cold and pain showed no marked impairment.

Course.—The patient died suddenly, about six months after the onset. The weakness in the limbs became less, but there was persistent paresis of the right motor fifth, and of the right sixth and seventh nerves. Disturbance of sensation became less, but there was always some impairment of the sense of position and movement on the left side, and ataxia of the left limbs.

Throughout the course of the disease, however, the patient complained of numbness in the left arm, leg, chest and abdomen. This persisted although disturbance of other forms of sensibility became less marked. This was the symptom of which the patient complained most.

COMMENT

There is no doubt that this patient had a small vascular lesion in the right side of the pons. The diagnosis of a tumor was never considered, because of the absence of any choking of the disks and of signs of increased intracranial pressure. Besides, the symptoms came on suddenly and became less as time went on, a course that would be expected in a vascular lesion.

The symptoms and location of the lesion are similar to those in Case 2, for, in both, deep sensibility was greatly impaired, and superficial sensibility was only slightly disturbed. The lesion unquestionably involved the region of the medial lemniscus, there being no implication of the posterior longitudinal bundle and only slight impairment of the motor fibers.

REPORT OF CASE WITH NECROPSY

Case 2.—*Clinical History.*—A married woman, aged 54, was admitted to the Polyclinic Hospital March 13, 1922, complaining of headache, vomiting, failing vision, numbness, and pain in the entire left side of the body and in the right side of the face, with weakness of the left side of the body. The symptoms began in May, 1921, with headache and dizziness, during which the patient always staggered forward. In a few weeks, this was followed by numbness in the left forearm and hand, which extended up the arm to the left side of the body, and afterward into the left leg. At the same time she began to be weak in the left side; and about three months later developed a twisting movement of the left hand and arm.

Six months after the onset, pain began in the entire left side of the body, first intermittently and later constantly. This pain was described by the patient as "burning, grinding, disagreeable and unbearable." She had noticed a diminution of tactile sense over the numb areas. The numbness and weakness came on simultaneously, and the patient stated that she had no strength in the left side of her body. During the two months previous to her entrance into the hospital, the neck had become rigid and she was disinclined to move the head.

Six weeks before admission, she complained of difficulty with vision, followed soon afterward by weakness of the right side of the face and jaw. In the course of a few weeks, she developed pain in the right side of the face. Internal

strabismus of the right eye had been noticed for three weeks, and the patient stated that she had suffered from diplopia for five or six weeks before coming to the hospital.

For four weeks she had noticed a "narrowing" of the throat and at times was unable to swallow coarse food; her jaws felt stiff, the gums felt hard, and she had the sensation of something dropping in the back of her throat.

During the summer of 1921 she had had spells of vomiting, which ceased after two weeks but recurred three months later. She had had from two to five vomiting attacks a day. The vomitus was brown, had no relation to food, and was projectile in character.

The patient had noticed progressive deafness in the right ear for a month before she was seen by us. Hearing in the left ear was fairly good, but below normal. There were no cardiovascular, renal or respiratory symptoms. Deglutition was difficult and food lodged between the teeth. The patient had lost 60 pounds (27 kg.) in two months.

Examination.—The head was normal in size and shape. The right eyeball was turned inward and there was paralysis of associated ocular movement to the right. The right external rectus was more paretic than the left internal rectus. There was hyperesthesia to pinprick in the distribution of the right fifth nerve, and hyperesthesia to all forms of sensation on the left side of the face, including the tongue and mucous membrane of the mouth. Sensitiveness to heat and cold was only slightly diminished on the right side of the face, but tactile sensation was almost lost.

With the mouth open, the lower jaw deviated to the right from paralysis of the motor fifth nerve. There were marked weakness of the right side of the face, peripheral in type, and almost total loss of hearing on the right side with possibly a little diminution in the left ear. There was weakness of the soft palate on the right. The tongue deviated slightly to the left.

Both extremities on the left side were weak and hypertonic. The biceps, triceps, patellar and Achilles reflexes were increased on the left, normal on the right. There was slight ankle clonus on the left. The plantar reflex was extensor on the left, flexor on the right. Sense of position and passive movement were markedly impaired on the left. There was astereognosis in the left hand. Heat, cold and tactile sensibility were greatly diminished on the left side of the body, and these stimuli caused a feeling of discomfort. Pin pricks, though sensibility was diminished, caused marked paresthesia on the left side. There was no difference in the disturbance of sensibility of the limbs between the proximal and distal parts. Station was fairly good, but in walking there was a tendency to stagger backward and to the left. There was no disturbance of superficial or deep sensibility of the trunk or of the extremities on the right side.

As has been mentioned, the patient had involuntary movements of the left arm and leg. These were not evident when she was quiet or asleep, but became obvious only on attempts to move the members; they were more marked in the arm than in the leg. They were described by her physician as searching movements, the index and ring fingers extended, and the others partially closed, with general rigidity of the whole hand, wrist and forearm. They bore a marked resemblance to athetoid movements. In testing the left limb for asynergy, it was found that, when the arm was extended from the shoulder, to and fro movements were normal so long as the patient kept her eyes on the limb. With the eyes closed, the movements were distinctly uncertain. With the eyes open, heel-to-knee tests were well performed, but

with the eyes shut, distinct ataxia was present, and the impression was obtained from these tests that these movements were not asynergic.

The neck was tender to touch posteriorly, more pronouncedly so on the left side. The anterior cervical glands were swollen. The heart and lungs were normal. The blood pressure was 112 systolic, 80 diastolic; the temperature and pulse were normal. The abdominal findings were negative. The blood count, Wassermann test and urine were normal.

Ocular Findings.—There was almost complete paralysis of the right external rectus muscle; the eyeball was constantly turned into the inner canthus and could be moved to the right only slightly. Inward rotation of the left eye was incomplete. Movements of the eyeballs up, down and to the left were normal. The right pupil was smaller than the left; both responded freely to light. In both eyes accommodation was good, and there was no hemianopia. There was no consensual response when light was thrown into the left eye, but there was slight response when light was thrown into the right eye.

The right optic disk was large, oval, and pale, with edges blurred. The veins were full but the arteries small. Paleness of the disk was pronounced on the temporal side. Elevation of the disk and of the fundus was about the same. The eye was myopic, the fundus being best seen with a lens of minus 7 diopters. The left disk was better defined, but the pallor was greater than in the right eye. The light projection field was good in all directions in both eyes. There was probably bilateral optic atrophy, but this was difficult to differentiate from change due to myopia.

Roentgen-Ray Examination.—There was no evidence of a neoplasm of the occipital bone, or of a lesion in the second, third, fourth or fifth cervical vertebrae.

MICROSCOPIC EXAMINATION OF SERIAL SECTIONS OF THE BRAIN STEM

The upper limit of the tumor (tuberculoma) was 1 cm. above the entrance of the fifth nerve. It gradually enlarged and reached its maximum in the lower part of the pons. It was confined to one side and in the greater part of its extent to the right tegmentum. It extended mediad to the raphe, laterad almost to the periphery, and posteriorly to within a millimeter of the floor of the fourth ventricle. Anteriorly it reached the deep pontile fibers of the brachium pontis, but at the level of the fifth motor nucleus it extended into the base of the pons, destroying most of the deep fibers of the middle cerebellar peduncle, and grazing the posterior fibers of the pyramidal bundles. It extended anteriorly to the pyramidal bundles and posteriorly pushed up the ependymal floor of the fourth ventricle. The tumor gradually disappeared in the upper part of the medulla, the last remnant being anterior and lateral to the inferior olive.

On the left side of the cerebellum, at the upper level of the medulla, there was a small tuberculoma involving the left tonsil and biventral lobule. It compressed the restiform body, with resultant loss of many of its nerve fibers. There was a pronounced tuberculous meningitis in this area, which in places had invaded the adjacent medulla for a millimeter, but had not destroyed any of the posterior column nuclei. There was a mild grade tuberculous meningitis about the entire brain stem, most marked about the posterolateral sulci of the medulla; meningitis was pronounced about the right seventh, eighth, ninth, and tenth nerves and both twelfth nerves with resultant slight nerve degeneration.

The following structures were destroyed by the pontile tumor: the medial and lateral lemnisci; most of the main sensory nucleus of the fifth nerve; the

spinal root of the fifth in the upper part of the medulla; the motor nucleus of the fifth nerve; the anterior part of the right superior cerebellar peduncle; the posterior longitudinal bundle; the seventh nerve and nucleus; most of the sixth nucleus and all of the sixth nerve; the middle cerebellar peduncle; and the posterior fibers of the pyramidal bundles.

At the upper part of the pons, all structures were normal except on the right where there was ascending degeneration of the afferent fibers of the medial and lateral lemniscus, and probably of some of the fibers of the spino-

Fig. 1.—Upper limit of tumor on right side of tegmentum of the pons.

thalamic tract; this latter tract is supposed to lie at the lateral part of the medial lemniscus, to which the tumor extended.

Frontal serial sections were made through the entire basal ganglions and stained by the Weigert hematoxylin method. A few hematoxylin and eosin sections were likewise made. The thalamus, corpus subthalamicum, substantia nigra, nucleus ruber, internal capsule and pes pedunculi, as well as the caudate and lenticular nuclei were carefully examined under the microscope and no lesion was found. The cells and fibers in these places were normal. The cyto-architecture of the cerebral and cerebellar cortices was normal.

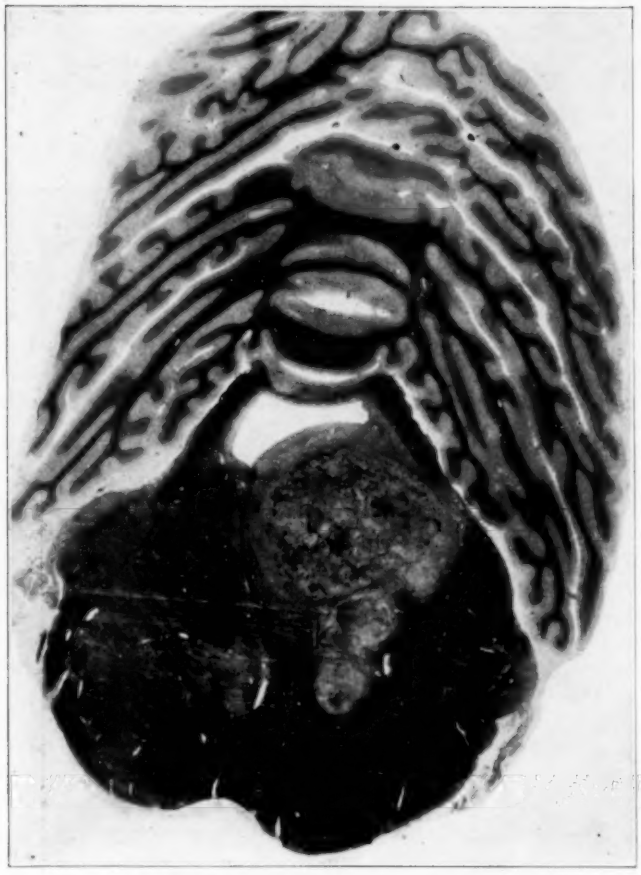

Fig. 2.—Tumor at level of the fifth nerve.

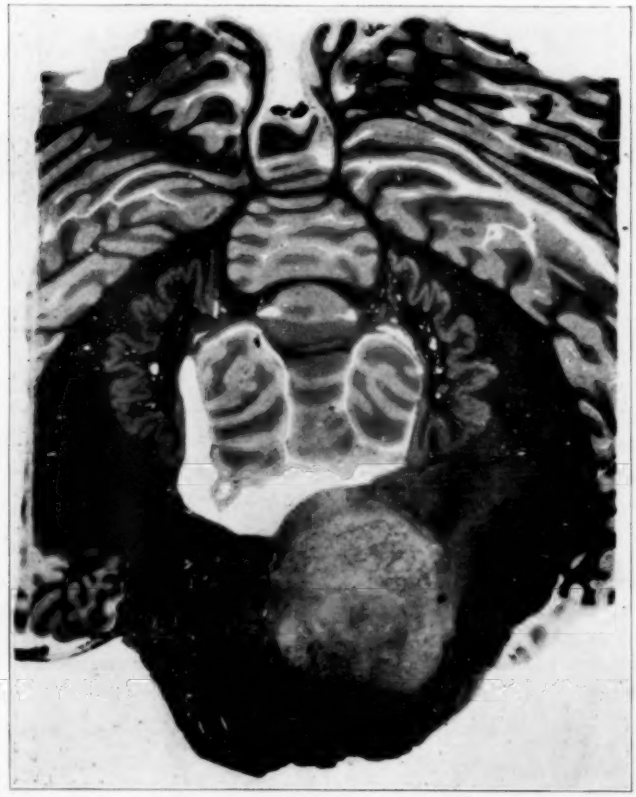

Fig. 3.—Lower level of the pons.

Fig. 4.—Tumor on left side of cerebellum compressing the restiform body.

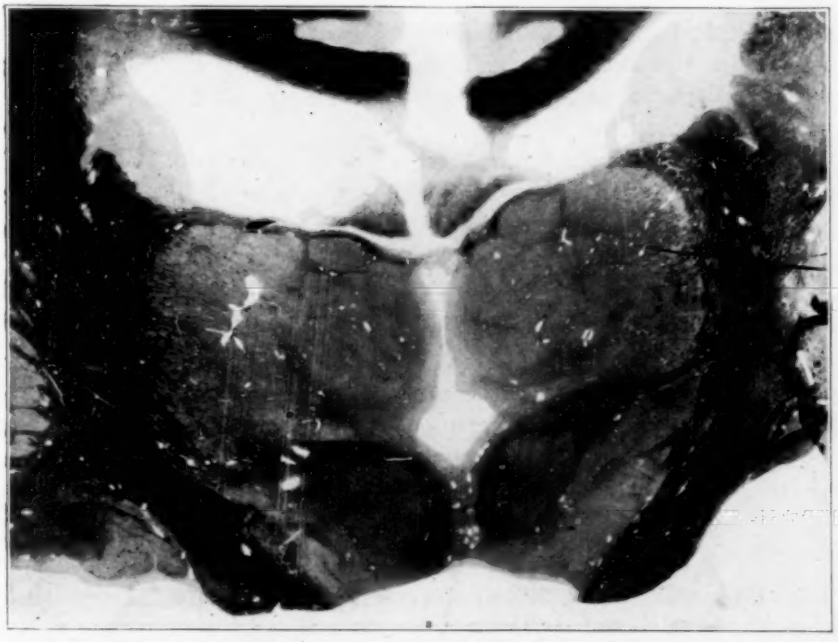

Fig. 5.—Frontal section of thalamus and subthalamic structures, all of which are normal.

COMMENT

That these two cases of pontile lesions produced symptoms of thalamic disease is noteworthy. The disturbance of deep sensibility, astereognosis, and ataxia are accounted for by destruction of the medial lemniscus. Inasmuch as the tumor in Case 2 extended laterad close to the periphery, and assuming the pathologic process reached the same area in Case 1, there is no doubt that some fibers of the spinothalamic tract were also involved.

Obviously, not every lesion of the pons can cause central pain. It is only when there is partial interruption of the sensory tracts, especially those of pain and temperature, that such a result is possible. Economo [11] stated that central pain might result from partial or total destruction of the fibers running in the spinothalamic and spinotectal tracts. In his case, in which the patient had severe pain, there was complete anesthesia to pain and temperature. This is contrary to the findings in the two cases reported here, as well as in Holmes' spinal cord lesions, in cases of pain due to vascular lesions, and in syringomyelia. In all these instances, it was found that sensibility either to pain, to heat or to cold was present, perhaps diminished, even when one or two of these three forms of sensations was totally lost. While we are at a loss to explain the pain in Economo's case, with total loss of superficial sensation, it is barely possible that there might have been a slight degree of sensitiveness to pain, heat, or cold retained. We assume that the brain cannot interpret pain unless some fibers of the spinothalamic and spinotectal tracts are intact. In all cases of pain due to thalamic disease, there is always some retention of sensibility to pain or temperature.

Holmes has shown that, as a result of the irritation of the central end of the divided pain conducting fibers, it may be possible to have central pain for a short time, even when there is total loss of sensibility from traumatic injury to the spinal cord.

CONCLUSION

Central pain can result from lesions of the sensory fibers in any portion of the intramedullary course from the spinal cord to the sensory area of the cortex. Pain can only occur if the nature of the lesion is such as to irritate these fibers, and not to block conduction through them.

DISCUSSION

ON PAPERS OF DRS. SPILLER, AND WEISENBURG AND STACK

DR. CHARLES K. MILLS, Philadelphia: I have been doubtful for some time as to the full validity of the Dejerine, Roussy, and Head interpretation of the thalamic syndrome. I have had cases of subthalamic lesion in which central pain was present, pain not explicable by irritation of the sensory roots. I

have had also a case with necropsy in which the thalamic syndrome was present from a lesion distinctly above the thalamus, the case of a growth involving the parietal lobe between the thalamus and the cortex, and not implicating the thalamus. The patient had the typical dysesthesia on which Head and Holmes have laid so much stress. Head especially has done much to simplify and clarify our knowledge of the symptomatology of the thalamus. He particularly has helped us to eliminate the idea of a sensory pathway around the thalamus to the cortex.

Dr. Foster Kennedy, New York: We have had two cases under observation at Bellevue Hospital. One was an aviator who in a nose-dive dropped four hundred feet and fractured his atlas. There occurred a temporary quadriplegia and a remarkable recovery. I saw him a year and a half later, when his sole complaint was terrific pain on both sides of his face, undoubtedly due to an involvement of the descending sensory nucleus of the fifth nerve. His teeth were on edge all the time and he had sharp pain in the distribution of the fifth nerve on both sides.

Another case was one of hematomyelia at the level of the first dorsal segment producing a hemiplegia which improved. Fifteen months afterward, there developed intense hyperesthesia on the left side corresponding to the first to the fifth dorsal segments. We thought that the hematomyelia had given rise to gliosis irritating the intramedullary tracts.

Dr. La Salle Archambault, Albany, N. Y.: The occurrence of pain in syringomyelia has of course been known for a long time. Not many years ago it was assumed that in most cases of syringomyelia there was a coexisting pachymeningitis, thus providing an easy explanation for the symptom. I am glad that Dr. Spiller has emphasized the possible rôle of involvement of the intramedullary sensory tracts in the genesis of so-called central pain. A most instructive case was reported recently by Drs. Foix, Thévenard and Nicolesco (*Revue neurologique* **20**:990, 1922). Their patient had had for many months paroxysmal excruciating pain in the distribution of the right trigeminal nerve, which was at first considered to be ordinary trigeminal neuralgia. Various injections and operations were performed without appreciable benefit, and none of the many medicinal agents employed seemed to afford him the slightest relief. The unfortunate man subsequently developed an absolutely analogous type of pain in the domain of the left trigeminal nerve, and eventually died of an old pulmonary tuberculosis. At the necropsy, to the surprise of all who had closely studied his case, two small cavities were found in the bulb, one on either side, occupying practically symmetrical areas within the nuclei of the descending roots of the fifth nerve.

Dr. George W. Hall, Chicago: I have a case at the present time of occlusion of the posterior inferior cerebellar artery, in which the patient complains of considerable pain, more or less paroxysmal, on the affected side. I should like to ask Dr. Spiller what his experience has been in regard to pain on the affected side.

Dr. Spiller, in closing: In my paper on occlusion of the posterior inferior cerebellar artery, I referred to the occurrence of pain.

SPASTIC PARAPLEGIA IN FLEXION

A CASE DUE TO A MENINGEAL TUMOR COMPRESSING THE LOWER CERVICAL CORD ON THE ANTERIOR AND RIGHT LATERAL ASPECTS *

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AND

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SAN FRANCISCO

The literature on spinal tumors has been enriched by the recent American contributions of Elsberg and Stookey, Frazier and Spiller, Abrahamson and Grossman, and by Adson. Refinements in diagnostic procedures and recognition of unusual clinical pictures have greatly aided diagnosis. The paucity of sensory symptoms, for example, in tumors situated on the anterior and anterolateral aspects of the cord has lately been emphasized. The case to be reported is one of spinal tumor in such location, but has the additional interest of presenting a flexion paraplegia, an unusual picture in spinal tumors. In a series of sixteen personal cases of verified spinal tumors affecting the cord the writer has met with but this one example.

A critical discussion and extensive review of the subject, with full literature references, will be found in the recent article of Babinski.[1] To Babinski belongs the credit of sharply differentiating the symptomatology of this form of paraplegia, spasmodic flexion paraplegia—"type cutanéo-réflex"—from the spastic extension paraplegia—"type tendino-réflex." It differs from the latter type in the contraction and often contracture of the limb segments in flexion, in the high grade of paresis, the activity of the defense movements, and particularly in the behavior of the reflexes—the tendon reflexes are normal, reduced, or even absent; the Babinski sign is usually present. The lessened activity of the tendon reflexes is found in a state of general anesthesia and is therefore not masked by increase of muscular tonus.

The pathologic conditions that most frequently give rise to this condition are said by Babinski to be diffuse spinal sclerosis, compression of the cord or bulb by a tumor or pachymeningitis, and bilateral lesions of the cerebral hemispheres. The mechanism is yet but

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* Read before the Forty-Ninth Annual Meeting of the American Neurological Association, Boston, June 1, 1923.

1. Babinski, John: Réflexes de défense, Rev. neurol. **38**:1049 (Aug.) 1922.

imperfectly understood. Anatomically, secondary degeneration of the pyramidal tracts is either absent or slight (Babinski). It is suggested that spinal irritation may play a rôle. There appears to be confusion in the identification of the syndrome of flexion paraplegia described respectively by French and English writers. To the latter, the condition under discussion is also evidenced by reflex movements with a divided cord and is not limited to a rigidity and contracture in flexion. It denotes a liberation of the cord from the influence of the higher centers. The difference is one of degree. In addition to Babinski, Walshe and Riddock particularly have discussed this question, as have Marie and Foix. The last word has not been said on the subject as well as on the subject of reflex cord action in general.

REPORT OF CASE

Clinical History.—A. R., a housewife, aged 55, a native of Guatemala, had complained of rheumatism and stiffness of both lower extremities beginning several months before November, 1919. At that time, evidently suspecting spinal cord disease, her attending physician obtained the cerebrospinal fluid, which showed an increase in globulins, a leukocyte cell count of 7, but a negative Wassermann reaction. The general blood picture was negative, showing no leukocytosis nor evidence of anemia.

The patient first came under the observation of the writer on April 23, 1920, referred by Dr. E. M. Aldana of San Francisco. The paresis of the lower extremities had progressed in the interval to a condition of marked paralysis. There was complaint of numbness and some weakness in the hands, and when closely questioned it was determined that the so-called "rheumatism" in the lower extremities was more numbness than true pain. No history of root pain or girdle sensation was obtained. The patient spoke no English and was communicated with through an interpreter. She was often irritable and emotional, and the examination was made difficult and complicated in all tests which demanded intelligent cooperation.

Difficulty was experienced in starting the flow of urine six weeks previously; then followed occasional incontinence of urine and feces, which progressed to marked urinary incontinence and obstinate constipation. From the onset there was great muscle tenderness in the lower limbs on pressure. Nothing of importance was obtained from inquiry into the hereditary, familial, or past history of the patient. Trauma to the spine, and hereditary or familial history of tumor were denied.

Physical Examination.—The patient, a well nourished woman in apparently good general condition, was confined to bed by her paralysis. The pulse was rapid at times—as high as 100 per minute. She was repeatedly examined prior to her operation on June 23rd. The cranial nerves showed no noteworthy changes. The hands showed a bilateral atrophy of the dorsal interossei and hypothenar muscles, more marked in the left hand. The hand-grips registered 10 kg. on each side. A slight hyperesthesia was found on the ulnar side of the right upper extremity, and a slight hypoesthesia on the corresponding side of the left upper extremity. The upper tendon reflexes showed no noteworthy changes. There were no vasomotor, trophic or tonus changes in the upper extremities, nor were there abnormal postures

Motor Phenomena.—The patient lay with the thighs flexed on the abdomen forming an angle of about 135 degrees; the legs were flexed on the thighs at approximately 90 degrees. The lower extremities were pressed together by contraction of the adductors but could be forcibly separated. The muscular tonus of the flexors was notably increased. The thighs could be passively extended further on the pelvis, but the legs were practically fixed at the knees. There was but slight, if any, active motion possible in any segment of the extremities owing to the spastic contracture of the flexors and adductors. The patellar and ankle jerks were markedly hypoactive, but varied in intensity in the different examinations. Of the skin reflexes, the Babinski sign was positive at first, but in the last examination made before operation it was negative. The Oppenheim, Mendel-Bechterew, Schaefer and Rosselimo signs were constantly negative. The abdominal reflexes were absent. Reflex movements of defense were constantly present on stimulation of both plantar and dorsal aspects of the foot. These were not very active, not of wide range, nor sustained. There was no clonus of the foot or patella.

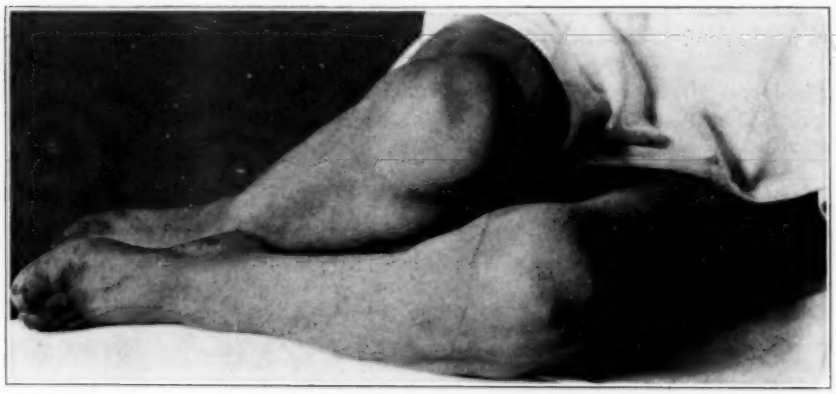

Fig. 1.—Flexion paraplegia.

Sensory Phenomena.—Light touch with cotton fiber was practically intact. Temperature was recognized as such below the segmental skin level of the second dorsal root, but hot and cold were confused. Pain tested by pin-prick was also disturbed below the second dorsal level and was either absent or greatly lessened. Deep sensation was apparently not disturbed for deep pressure, toe positions, vibration sense, localization or determination of compass points. Tenderness on percussion over the vertebra prominens was constant.

Laboratory Findings.—A lumbar puncture was performed on May 5. The pressure of the fluid was 60 mm. The fluid was of a striking yellow color; 9 c.c. were withdrawn and replaced by 7 syringefuls of air (ordinary hypodermic syringe). Subsequent roentgenograms of the spine failed to demonstrate any demarcation level due to block. Previous complete roentgen-ray examination of the spine had failed to reveal any pathology. The analysis of the cerebrospinal fluid showed 3 leukocytes per cubic millimeter, and a marked globulin reaction. The Wassermann reaction was negative in the fluid as well as in the blood. The routine laboratory work on the blood and urine was negative.

A diagnosis having been made of a tumor compressing the cord in the lower cervical region, an operation was advised. This was performed by Dr. Philip K. Gilman, June 23, 1920.

Operation.—The laminae of the fifth, sixth and seventh cervical vertebrae were removed. Pulsation of the dura was absent below the level of the fifth cervical body. On opening the dura a purplish-brown tumor was seen lying to the right and in front of the cervical cord and extending from about the middle of the fifth lamina downward to a little beyond the seventh lamina. This tumor measured approximately five centimeters in length by more than one centimeter in diameter, encroached on the spinal canal more than one-half its diameter, and compressed the cord in this region quite firmly toward the left. Toward the lower pole of this cylindrical mass the posterior roots of apparently the first dorsal nerve crossed the tumor; the posterior roots of

Fig. 2.—The tumor exposed.

the segment above crossed the tumor toward its upper rounded end. The tumor was held rather firmly in place first, by a fairly dense attachment to the inner surface of the dura opposite the lamina of the sixth cervical vertebra, and secondly, by a thin almost transparent tough membrane which was probably the distorted dentate ligament. After elevating the posterior branches that coursed across the tumor, the enucleation of the growth was begun at the lower pole, but it was necessary to cut the transparent structure identified as the dentate ligament and carefully to separate the growth from the dura before the lower half of the tumor could be raised from its bed. The upper pole was approached in the same way and after retracting upward the nerve fibers which coursed across it, the tumor was delivered. On elevating the tumor at this stage a second large lobe was brought to view extending around to the front of the cord. After retracting the cord itself towards the left and using a blunt dissector apparently the entire tumor was delivered and removed in one piece.

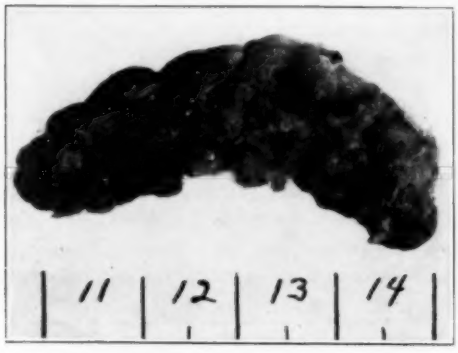

Fig. 3.—Dural endothelioma measuring 4.75 cm. in length.

Fig. 4.—Four months after removal of tumor; patient able to stand and walk with assistance.

Pathologic Examination of Tumor (Dr. F. E. Blaisdell).—The specimen consisted of a mass of tissue measuring 4.75 cm. in length, 1 cm. in width and a little less in thickness. It was elongately oblong in form, and more or less irregular on the surface from slight lobulation. It was rather soft in consistency and gritty when rubbed between the fingers (psammoma); the color was reddish gray. The form may also be said to have been flattened. Microscopic examination of paraffin sections revealed many concentric masses of cells and numerous calcareous granules. Many of the whorls showed hyaline degeneration of the collagen. The tumor cells were rather large, with large oval nucleus without much chromatin; those caught on edge in the section appeared flat; many of them were arranged concentrically in groups, with or without collagen fibrils between them. There was a very moderate amount of stroma, with many small blood vessels. The calcareous granules showed a concentric lamellar structure—lines of growth from calcification of the hyaline substance. Type of cell: endothelium. Diagnosis: Endothelioma, dural.

Postoperative Course.—After the operation there was a period of mental confusion which began to clear on July 9. The position and function of the legs on this date were approximately the same as before operation, with the exception that the left leg could be passively extended on the thigh, whereas the right knee seemed fixed by contracture. As this condition had not ameliorated, on August 10, an examination under anesthesia was made by Dr. Gilman who found by gentle extension that the left leg could be extended to within 25 degrees of full extension, and the right somewhat less. Both legs were straightened out, the right with some difficulty because of fairly dense adhesions in the joint and about it. Both extremities were put in plaster casts.

The patient was treated by mechanotherapy and physiotherapy until October 14, prior to her departure for home. She had been fitted with braces and with these could stand and walk with assistance. Voluntary motion was returning in the left lower extremity, but was much retarded in the right, due to flexor and adductor spasm. Sphincter control was regained and sensation was returning. The inferior tendon reflexes had become fairly active and the Babinski reflex was definitely elicited on both sides.

No definite conclusions can be drawn as to the mechanism of the flexion paraplegia which occurred in this case. The location, localization and extent of the tumor may one or all have been factors in its production. The fact that slight, if any, changes have been observed in the pyramidal tracts in similar cases, might suggest that the location of the tumor in front of the dentate ligament may have spared the lateral pyramidal tracts from degeneration, but may have involved the extrapyramidal long fiber tracts, namely, the vestibulospinal and tectospinal tracts, which descend in the anterior white matter of the cord between the anterior horn and the periphery.

DISCUSSION

Dr. Ramsay Hunt, New York: The distinction which Dr. Schaller tries to draw is good and a step in the right direction. We know that a lesion of the internal capsule produces a certain type of hemiplegia, with a corresponding

type of contracture; that a lesion of the corpus striatum (or of the pallidal system) produces a unilateral hemiplegia of the paralysis agitans type, with its corresponding rigidity.

In practice, hemiplegia is usually a combination of these two factors. In other words, as I have termed it, it is a pallidopyramidal type of hemiplegia, and the curious dissimilarities and the different degrees of rigidity and deformity in hemiplegic disturbance correspond to different degrees of involvement of these two mechanisms, the paleokinetic and the neokinetic, or as Dr. Wilson has termed them, the old and the new motor pathways.

Dr. Schaller is attacking this problem from the spinal cord. I believe that a lesion of the pyramidal tract alone, as we see it in primary spastic paraplegia, produces a paraplegia of extension, and that the paraplegia of flexion follows a more general compression of the spinal cord. Whether or no he is correct in the assumption that it is due to involvement of the extrapyramidal tracts alone, I think is an interesting question.

Dr. Charles Elsberg, New York: We have seen a number of patients with spinal cord tumors with paraplegia of the flexion type. The tumors have been on the ventral or ventrolateral aspect of the cord, if they were intradural. Quite frequently we have seen the flexion type with extradural tumors which are, of course, very frequent on the anterior aspect, or mostly on the anterior aspect. But we have seen paraplegia in flexion most often in the last stages of spinal compression. The last stage of spinal compression is usually paraplegia of the flexion type. I do not think that freedom from degeneration in the spinal system, such as described by Dr. Schaller, would be sufficient to explain this paraplegia in flexion.

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A STUDY OF BLADDER AND RECTAL DISTURBANCES IN SPINAL CORD TUMORS*

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NEW YORK

While reviewing this series of spinal cord tumors a few years ago with Dr. Elsberg, we were surprised to find that bladder and rectal disturbances were relatively late in their appearance; not infrequently occurring only a few days or weeks before operation. In this study an effort is made to determine if bladder or rectal disturbances are more common in any one group of spinal cord tumors, such as intramedullary, extramedullary or extradural tumors, etc., and if in any of these groups the time occurrence of bladder or rectal disturbances differs.

Chart 1 shows the relative frequency of bladder and rectal disturbances in the various groups of spinal cord tumors. It will be seen that bladder and rectal disturbances occur in 41 per cent. of the cases in intramedullary tumors, 78 per cent. in extradural, 80 per cent. in extramedullary and 83 per cent. in tumors of the conus and cauda equina. Thus bladder and rectal disturbances are twice as frequent in extramedullary, extradural and tumors of the conus and cauda equina as in intramedullary growths.

The relative frequency of each type of bladder and rectal disturbances is shown in Table 1:

TABLE 1.—*Relative Frequency of Bladder and Rectal Disturbances*

	Extramedullary, Percentage	Intramedullary, Percentage	Extradural, Percentage	Conus and Cauda, Percentage	Average Percentage
Relative frequency of bladder incontinence	45	25	42	66	46.5
Relative frequency of dysuria	11	16	35	16	19.5
Relative frequency of rectal incontinence	31	8	7	16	15.5
Relative frequency of obstinate constipation	24	40	49	48	42.5

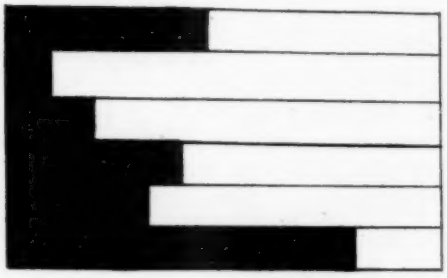
Bladder incontinence thus occurred most frequently in tumors of the conus and cauda. It was nearly twice as frequent in extramedullary and extradural tumors (45 per cent.) as in intramedullary tumors (25 per cent.). Rectal incontinence, on the other hand, occurred twice as frequently in extramedullary as in tumors of the conus and cauda, and four times as frequently as in intramedullary or extradural tumors. Dysuria occurred in about the same frequency in all groups with the exception of extradural tumors, in which dysuria occurred more than twice as frequently. Obstinate constipation was a rather common com-

* Read by title before the Forty-Ninth Annual Meeting of the American Neurological Association, Boston, June 1, 1923.

I. Intramedullary Spinal Cord Tumors

		Per cent.
Total number of cases	12	
Bladder incontinence	3	25
Urgency	2	17
Rectal incontinence	1	8
Obstinate constipation	5	41
Total number of cases showing bladder disturbances or rectal or both		41

II. Extramedullary Spinal Cord Tumors.

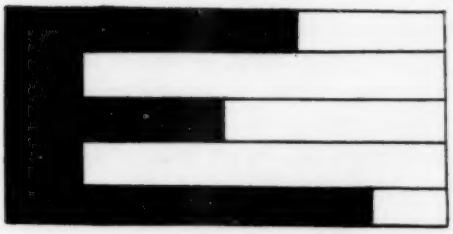


Total number of cases	42	
Bladder incontinence	19	45
Dysuria	5	10
Urgency	8	19
Obstinate constipation	10	24
Rectal incontinence	13	31
Total number of cases showing bladder disturbances or rectal or both		80

III. Extradural Spinal Cord Tumors.

Total number of cases	14	
Bladder incontinence	6	43
Dysuria	5	35
Obstinate constipation	7	50
Rectal incontinence	1	7
Total number of cases showing bladder disturbances or rectal or both		78

IV. Tumors of Conus and Cauda Equina.



Total number of cases	12	
Bladder incontinence	8	66
Dysuria	2	17
Obstinate constipation	6	50
Rectal incontinence	2	17
Total number of cases showing bladder disturbances or rectal or both		83

Chart 1.—Relative Frequency of Bladder and Rectal Disturbances.

plaint in all, occurring with about the same relative frequency. It is a sign of little value, since it is often difficult to differentiate the rather habitual marked constipation of which many of the patients complained from the obstinate constipation due to a spinal cord neoplasm.

In order to determine the average time in the disease at which bladder or rectal symptoms appeared, the period from the presenting symptom to operation was taken as 100; the time at which bladder or rectal signs appeared was figured accordingly, and expressed in terms of percentage of time duration. In all but two of the cases in the entire series, bladder and rectal symptoms did not appear until well into the latter half of the course of the disease. In these two exceptions, bladder and rectal signs were present at the onset and appeared as the presenting symptom. More will be said of these cases later.

Bladder incontinence and rectal incontinence occurred earliest in conus and cauda equina tumors, when approximately 60 per cent. of

TABLE 2.—*Average Time Duration of Disease from Presenting Symptom to Appearance of Bladder Incontinence*

	Extramedullary, Percentage	Intramedullary, Percentage	Extradural, Percentage	Conus and Cauda, Percentage	Average Percentage
From presenting symptom to appearance of bladder incontinence	74	85	88	57	76
From presenting symptom to appearance of rectal incontinence	68	80	94	63	76
From presenting symptom to appearance of obstinate constipation	78	87	72	52	72

the time duration of the disease had passed. Extramedullary tumors were next to show vesical and sphincter disturbances, with intramedullary and extradural tumors showing these signs extremely late. Particularly in the latter, vesical incontinence did not occur until more than 90 per cent. of the time duration of the disease had passed. The figures are indicated in Chart 2 and are summarized in Table 2.

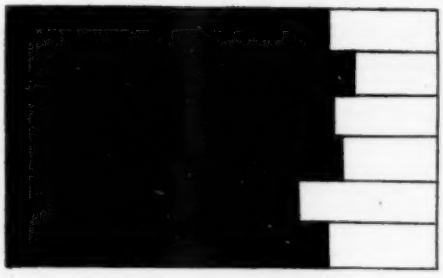
It was thought that probably there might be some relationship between tumors situated at various segmental levels and the appearance of sphincteric disturbances. The segmental distribution of the various tumors in intramedullary, extramedullary and extradural groups may be seen in the accompanying illustration. Unfortunately, tumors of the conus and cauda do not permit an accurate localization at operation, since many nerve roots are involved and identification of the individual roots is often impossible.

The tumors which gave rise to bladder or rectal disturbances are indicated by straight lines, while those in which such disturbances did not occur are represented by broken lines.

Sphincteric disturbances were found in a great variety of tumor distribution, and did not seem to show any particular relationship to

I. Tumors of Conus and Cauda Equina.

II. Extramedullary Spinal Cord Tumors.

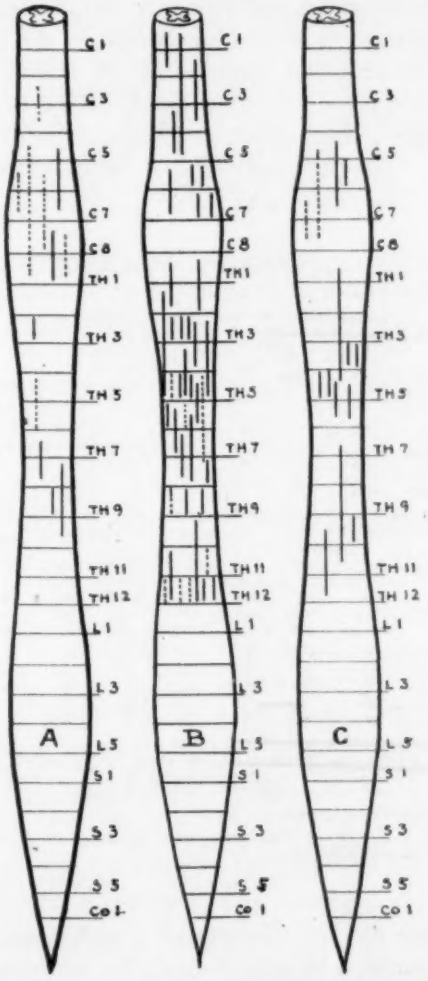


III. Intramedullary Spinal Cord Tumors.

IV. Extradural Spinal Cord Tumors

Chart 2.—Average time duration of disease from presenting symptom to appearance of bladder and rectal disturbances.

the segmental level involved. Of tumors at the same segmental level, some gave rise to sphincteric disturbances and others did not. For example, this was true in such widely separated regions as the fifth and sixth, and eleventh and twelfth thoracic segments in extramedullary



Segmental distribution of spinal cord tumors: *A*, intramedullary; *B*, extramedullary; and *C*, extradural. Bladder or rectal disturbances were present in all except those indicated by dotted lines. It was impossible to figure accurately the segmental level of tumors of the conus and cauda equina, hence these are not illustrated.

tumors, or in the fifth to eighth cervical segments in intramedullary growths. This would seem to indicate that there is no particular segment which is especially concerned with sphincter control either of the afferent or efferent arc.

RELATION OF SPHINCTERIC DISTURBANCE TO PRESSURE ON THE WHITE COLUMNS OF THE SPINAL CORD

Since, at the levels concerned in this series, sphincter disturbances appeared to be independent of segmental distribution, with the exception of tumors of the conus and cauda, it was considered possible that tumors situated ventral or dorsal or lateral to the spinal cord might give rise to bladder or rectal disturbances by reason of their pressure on the ventral, dorsal or lateral columns in particular. From this viewpoint, only the extramedullary and extradural tumors could be studied, since the precise position and limits of an intramedullary glioma could rarely be determined.

TABLE 3.—*Extramedullary Spinal Cord Tumors*

A. Relation of Tumors to Spinal Cord

Position	No. of Cases	Percentage*	
Ventral	11	26	Ventral and ventrolateral tumors, 33 per cent.
Ventrolateral	3	7	
Dorsal	22	52	Dorsal and dorsolateral tumors, 59 per cent.
Dorsolateral	3	7	
Lateral	3	7	

B. Relation of Tumors to Spinal Cord and Appearance of Bladder and Rectal Disturbances

	Bladder Incontinence		Dysuria		Frequency		Rectal Incontinence		Constipation	
Position	No. of Cases	Percentage*	No. of Cases	Percentage*	No. of Cases	Percentage*	No. of Cases	Percentage*	No. of Cases	Percentage*
Ventral	5	27	2	40	1	11	4	33	3	30
Ventrolateral	2	11	0	0	1	11	0	0	0	0
Dorsal	7	38	2	40	6	66	5	41	5	50
Dorsolateral	3	16	0	0	0	0	2	16	1	10
Lateral	1	5.5	1	20	1	11	1	8.3	1	10

Summary: 38 per cent. of bladder incontinence occurred in ventral and ventrolateral tumors.
55 per cent. of bladder incontinence occurred in dorsal and dorsolateral tumors.
22 per cent. of rectal incontinence occurred in ventral and ventrolateral tumors.
38 per cent. of rectal incontinence occurred in dorsal and dorsolateral tumors.

* Total number of each group (bladder incontinence, etc.) is taken as a unit and percentages are figured accordingly.

For purpose of diagnosis and description, tumors in this series have been classified by Dr. Elsberg as ventral, ventrolateral, lateral, dorsolateral and dorsal. Ventral tumors lie between the two ventral roots, dorsal tumors between the two dorsal roots and lateral tumors between the ventral and dorsal roots. Those in front of the dentate ligaments are called ventrolateral and those behind the dentate ligaments are classified as dorsolateral..

It will be seen from a study of Tables 3 and 4 that approximately 60 per cent. of all extramedullary tumors are dorsal and dorsolateral, and 36 per cent. ventral and ventrolateral, and that approximately the same proportions are maintained in regard to the frequency with which bladder and rectal incontinence were found.

As regards extradural tumors, the condition was quite the reverse—there being three times as many ventral and ventrolateral tumors as dorsal and dorsolateral tumors. In this group it is interesting to note that in 100 per cent. of the cases showing bladder or rectal incontinence the tumor was ventrally situated, while in 60 per cent. of those showing dysuria it was dorsally placed. It is possible that this is a coincidence, since this relationship of the ventral columns to incontinence was not confirmed by a study of the extramedullary tumors. Had there been any particular significance in pressure on the ventral columns as a cause of vesical or rectal disturbances, this relationship would have been found to be constant throughout the series. But this was not true.

TABLE 4.—*Extradural Spinal Cord Tumors*

A. Relation of Tumor to Spinal Cord

Position	No. of Cases	Percentage	
Ventral	7	49	Ventral and ventrolateral tumors, 63 per cent.
Ventrolateral	2	14	
Dorsal	2	14	Dorsal and dorsolateral tumors, 21 per cent.
Dorsolateral	1	7.1	
Lateral	2	14	

B. Relation of Tumor to Spinal Cord and Appearance of Bladder and Rectal Disturbances

	Bladder Incontinence		Dysuria		Rectal Incontinence		Constipation	
Position	No. of Cases	Percentage*	No. of Cases	Percentage*	No. of Cases	Percentage*	No. of Cases	Percentage*
Ventral	5	83	1	20	1	100	5	83
Ventrolateral	1	17	0	0	0	0	1	17
Dorsal	0	0	2	40	0	0	0	0
Dorsolateral	0	0	1	20	0	0	0	0
Lateral	0	0	1	20	0	0	0	0

Summary: 100 per cent. of incontinence occurred in ventral or ventrolateral tumors.
100 per cent. of cases showing rectal incontinence occurred in ventral tumors.
100 per cent. of cases showing obstinate constipation occurred in ventral or ventrolateral tumors.
60 per cent. of cases showing dysuria occurred in dorsal or dorsolateral tumors.

* Total number of each group (bladder incontinence, etc.) is taken as a unit and percentages are figured accordingly.

RELATION OF SPHINCTERIC DISTURBANCES TO VARIETY OF TUMOR

In studying the tumor varieties in their relation to the occurrence of sphincter disturbances, it was considered that the size and consistency of the various neoplasms concerned might be important. From this standpoint, the extramedullary intradural tumors form an interesting contrast to the extradural group.

The extramedullary intradural tumors were, in the order of their frequency, endothelioma, fibroma, neurofibroma and sarcoma. Other varieties occurred so infrequently as not to permit of deductions being drawn.

The average size of the endothelioma, fibroma and neurofibroma was very nearly the same, endothelioma averaging 3 x 2 cm., fibroma 2.3 x 1.3 cm., and neurofibroma 2.3 x 1.3 cm.

Bladder and rectal incontinence occurred comparatively early in the endotheliomas and sarcomas (approximately 60 per cent.), and late in fibromas and neurofibromas (80 per cent.).

TABLE 5.—*Extramedullary Spinal Cord Tumors*

A. Tumor Varieties and Their Relation to Bladder Incontinence

Variety	Consistency	Size	Percentage* Time Duration	
Fibroma	Firm	3 × 1	74	Average size, 2½ × 1
	Soft	2½ × 1	66	Average percentage
	Firm	2 × 1	80	time, 73
Neurofibroma	Firm	2½ × 1½	97	Average size, 2.3 × 1.3
	Firm	2½ × 1½	86	Average percentage
	Soft	2 × 1	69	time, 84
Endothelioma	Hard	3 × 1½	70	
	Hard	2½ × 1½	91	
	Hard	5 × 2	50	Average size, 3.3 × 1.6
	Hard	2 × 1	63	Average percentage
	Hard	3 × 1½	Onset	time, 62
	Hard	3 × 1½	75	
	Hard	5 × 2	88	
Sarcoma	Soft	5 × 1 (?)	66	Average size, 5.5 × 1
	Soft	6 × 1	70	Average percentage time, 68

B. Tumor Varieties and Their Relation to Rectal Incontinence

Fibroma	Firm	3 × 1	74	Average size, 2½ × 1
	Firm	2 × 1	80	Average percentage
	Soft	2½ × 1	66	time, 73
Neurofibroma	Firm	2½ × 1½	97	Average size, 2.3 × 1.3
	Firm	2½ × 1½	86	Average percentage
	Soft	2 × 1	69	time, 84
Endothelioma	Hard	3 × 1½	70	
	Hard	2½ × 1½	91	Average size, 3.1 × 1.5
	Hard	5 × 2	50	Average percentage
	Hard	2 × 1	63	time, 68
Sarcoma	Soft	5 × 1 (?)	66	

C. Tumor Varieties and Their Relation to Dysuria

Fibroma	Hard	2½ × 1	87	
Neurofibroma	0	0	0	
Endothelioma	Firm	2½ × 1	78	
	Firm	5 × 1½	80	Average size, 2.8 × 1.3
	Firm	2 × 1	87	Average percentage
	Firm	2 × 1	—	time, 81
Sarcoma	Soft	2 × 1	88	

The reason for the early appearance of bladder and rectal disturbances in both the relatively small endotheliomas and the rather large sarcomas may be due to the fact that these endotheliomas were often extremely hard. Quite a few had numerous psammoma bodies and consequently, though small, were hard. A small, yet hard, tumor would give rise to signs earlier than a tumor of the same size, but of less density. Certain fibromas and neurofibromas may be very hard, but in this group of tumors which gave rise to incontinence the endotheliomas appeared to be of greater density. Sarcomas may give rise

to early signs, even though soft, due to their very large size, being on the average more than twice as large as the fibromas or endotheliomas.

Thus it would seem that in extramedullary tumors the spinal cord shows evidence of compression and disturbances in its conduction paths early in small, hard tumors, and also in soft tumors when they have attained large size. In sarcomas a large size is reached rather early. The relations with sphincteric disturbance are given in Table 5.

Among the extradural tumors, tabulated in Table 6, the sarcomas predominate. They are soft and attain relatively large size, averaging 6 x 2 cm. Their presence is well borne by the spinal cord, and bladder and rectal disturbances occur extremely late, 90 per cent. of the disease duration having passed before such symptoms appear. Cer-

TABLE 6.—*Extradural Spinal Cord Tumors*

A. Tumor Varieties and Their Relation to Bladder Incontinence

Variety	Consistency	Size	Percentage Time Duration
Fibroma	Hard	—	72
Endothelioma	Hard	—	92
Sarcoma	Soft	9 × 2	85
	Soft	4 × 2	94
	Soft	Large	90
Tuberculoma	Soft	4½ × 1½	92

B. Tumor Varieties and Their Relation to Rectal Incontinence

Sarcoma	Soft	4 × 2	94

C. Tumor Varieties and Their Relation to Dysuria

Fibrosarcoma	Firm	4 × 1	96
	Soft	4½ × 1½	Onset
Sarcoma	Soft	3 × —	78
Chondroma	Hard	3 × 1	67
Angioma	Hard	5 × 1½	84

tain mechanical factors may be responsible for the latter. The sarcomas tend to spread along the length of the vertebral canal and frequently obtain additional space by causing pressure atrophy of the bone. The diameter of the vertebral canal in these cases is often remarkably increased. The dura with the cerebrospinal fluid, which forms an inelastic water bed beneath it, offers greater resistance to pressure and perhaps accounts for the early vague character of the symptoms and the late appearance of bladder and rectal disturbances in extradural tumors.

That the consistency of the tumor has much to do with the appearance of the symptoms, is shown by the fact that a chondroma, which is an extremely hard tumor, gave rise to sphincteric disturbances very early as compared with the other tumors in this extradural group.

Since bladder and rectal disturbances occur relatively early in those tumors which cause the most marked pressure effects on the spinal cord,

and since bladder and rectal disturbances usually appear extremely late, it was thought that bladder and rectal symptoms would not be found unless they were associated with marked signs of interruption of cord conductivity, either afferent or efferent. This aspect is considered in the following:

RELATION OF BLADDER AND RECTAL INCONTINENCE TO SENSORY CHANGES

(A). *Intramedullary Tumors.*—In each of the cases with bladder and rectal incontinence, marked sensory changes were present, amounting nearly to complete anesthesia and analgesia. Marked sensory changes were present in two cases without bladder or rectal disturbances. In one of these, the sacral dermatomes were uninvolved; and in the other, the main sensory changes were unilateral. Thus certain dermatomes were unaffected, an indication that conductivity of some afferent impulses within the cord—sufficient at least to maintain bladder and rectal function as well as to supply certain somatic areas—was still possible.

(B). *Extradural Tumors.*—In all the cases of this series in which bladder or rectal incontinence occurred, marked sensory changes were present, and in no case were marked sensory changes present without some bladder symptoms (frequently dysuria or retention). Dysuria occurred four times. In two of the cases the sensory signs were marked, and in two they were moderate, showing that if even restricted afferent impulses are possible, bladder incontinence does not appear.

(C). *Extramedullary Tumors.*—Bladder incontinence occurred in this series nineteen times. In all but two cases, marked sensory changes were present. In one case occasional incontinence with dysuria was present, yet no sensory changes were made out. However, immediately after lumbar puncture, incontinence occurred and marked level sensory changes were then found. The tumor was a typical lipoma, laterally placed, and at the level of the third thoracic segment. In another case moderate sensory changes were present, and incontinence occurred only when the patient was recumbent. When the patient was up and about he had full control of his bladder. The tumor, in this patient, was a firm endothelioma lying ventral to the tenth to twelfth thoracic segments. It is possible that when the patient was recumbent the tumor was so placed as to cause greater pressure on the cord than when standing.

With these two exceptions in no case in this series did bladder or rectal incontinence occur unless marked sensory changes were present. However, the converse was not true, since in one case marked somatic sensory changes were present without bladder or rectal signs.

RELATION OF BLADDER INCONTINENCE TO MOTOR CHANGES

(A) *Intramedullary Tumors.*—In two of the three cases of this group in which incontinence occurred marked sensory changes and marked motor changes were present, in one marked motor alone. Yet, in other intramedullary cases, marked motor changes without marked sensory signs were present, but no disturbances of any kind in bladder or rectal functions were found.

(B) *Extradural Tumors.*—Marked paralysis with only moderate sensory changes was present in two of the extradural group, yet neither bladder nor rectal symptoms were manifested.

(C) *Extramedullary Tumors.*—Bladder incontinence occurred in this series nineteen times. In fifteen of them, marked motor changes were present; in three only moderate motor changes with marked sensory signs; while in another no motor changes were present. Thus in four cases, incontinence occurred with only moderate motor changes or no motor changes at all, while in one case marked motor changes were present without bladder or rectal incontinence.

Rectal incontinence, with only moderate motor involvement, but with marked sensory changes, occurred in two cases. Thus there seems to be little relation between the extent of the motor involvement and the bladder and rectal disturbances—certainly not as definite a relation as is observed between bladder and rectal incontinence on the one hand, and sensory changes on the other.

Thus in the majority of cases the pressure of a tumor sufficient to disturb the somatic afferent impulses through the spinal cord will profoundly interfere with the passage of visceral impulses. However, in certain cases of this group neither the somatic motor nor somatic sensory were sufficiently involved to account in themselves for the occurrence of bladder or rectal incontinence. If it could be shown that the visceral afferent pathway within the spinal cord is separate from the somatic afferent pathway, such instances may be understood. In fishes, the primitive pain pathway consists of a number of short neurons with numerous synapses in the gray matter; the long neuron, spinothalamic tracts are more recent acquisitions. Karpus and Kreidl [1] have shown that pain stimuli are transmitted even after section of both halves of the cat's cord at different levels; probably by a similar pathway of short relays.

Davis [2] found that visceral afferent impulses producing pressor reflexes do not ascend by the same pathway as those conducting the somatic afferent impulses giving rise to the same reflex, but that the

1. Karpus and Kreidl: Pflüger's Arch. **158**:275, 1914.
2. Davis: Am. J. Physiol. **59**:321, 1922.

visceral afferent impulses were carried by relays of short spinal neurons with synapses in the gray matter.

If afferent visceral impulses thus have their separate pathway, it is possible to explain those cases in which bladder or rectal disturbances occurred without corresponding somatic afferent or efferent disturbances. Furthermore, since the visceral afferent path is one of short relays with numerous synapses at various levels in the gray matter, it may not be involved until a relatively late period, after other pathways have been markedly compressed. A pathway of short neurons and numerous relays in the gray matter is more resistant, and its conductivity is less apt to be interrupted, until after the greater part of long neuron conductivity has been severely compressed.

Of the various modalities of sensation the earliest to return, following the removal of a spinal cord neoplasm, are the visceral sensibilities as indicated by a consciousness of bladder fulness and the presence of an impulse to evacuate the bowels. In many instances the visceral impulses are the first to return, preceding those of pain and temperature, touch or vibration, etc. It is not infrequent to find that the return of visceral sensations has taken place within a few days after operation.

CONCLUSIONS

1. Bladder and rectal symptoms in spinal cord tumors occur twice as frequently in extramedullary, extradural and conus and cauda equina tumors as in intramedullary tumors.

2. Bladder and rectal symptoms do not take place until relatively late in the course of the disease, being earlier in tumors of the conus and cauda and extremely late in extramedullary, intramedullary and extradural tumors, in the order named.

3. In this series no particular incidence relation to any given segmental level above the twelfth thoracic segment could be determined.

4. No special significance was found in the relation of the appearance of bladder and rectal signs to the location of the tumor on the ventral, dorsal or lateral columns of the spinal cord, though a closer relation to the ventral aspect is suggested.

5. Bladder and rectal signs occurred irrespective of the pathology of the various tumors; but hard tumors, though small, gave rise to signs earlier than did softer tumors which were larger. When relatively very large, even though soft, bladder and rectal signs occurred earlier. This was particularly true of sarcomas.

6. When bladder or rectal incontinence occurred, marked sensory changes were present in the majority of cases of this series.

There seemed to be less relationship between bladder and rectal incontinence and the presence of marked motor changes than between bladder and rectal incontinence and sensory changes.

7. From the clinical side there is evidence to point to a visceral afferent pathway within the spinal cord distinct from the somatic afferent path.

8. The resistance of the visceral afferent pathway to pressure and its prompt return to function when pressure is relieved, suggest from a clinical standpoint, that it is a short neuron pathway with numerous synapses.

9. Usually bladder and rectal disturbances are the earliest functions to return after removal of a spinal cord neoplasm—preceding the long neuron paths of vibratory, muscle, joint, tendon, pain and temperature sensations. Thus the short neuron visceral afferent path with its numerous synapses regains its conductivity earlier.

VISUAL HALLUCINATIONS AS A CEREBRAL LOCALIZING PHENOMENON

WITH ESPECIAL REFERENCE TO THEIR OCCURRENCE IN TUMORS OF THE TEMPORAL LOBES *

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Visual hallucinatory phenomena in cases of definitely authenticated organic disease of the brain were recorded as early as 1879 when Westphal [1] cited such an instance and gave the clinical and necropsy findings. His case was in a man, aged 42, who suffered from focal epilepsy involving the left side. Before death he had developed a left hemiplegia and left homonymous hemianopia. His hallucinations consisted in seeing not only bright colors before his eyes, but on one occasion the appearance of "a sword hovering over his head, about to drop on him." At this time "he stared up at the ceiling as if he saw something horrible there." Postmortem, there was found "atrophy and softening of the posterior half of the right hemisphere."

Since this early reference, numerous instances not only of color phenomena, but also of the apparent seeing of figures and objects by patients who later were shown to have had tumors or other organic brain lesions have been reported. It was not until 1886, however, that an instance was published of a patient who had visual hallucinations projected in a single lateral direction, corresponding with an homonymous hemianopia toward the same side. In this year Seguin [2] recorded such an observation, and although the lesion was not verified, the history and clinical findings leave small doubt as to the organic nature of the process, which the author believed to be an embolus situated in the left occipital lobe.

Seguin's case was a woman, aged 34, who "at the close of her third confinement, just after the child was born, had a peculiar attack in which she experienced a 'snap,' or sudden pain in the left temple, and felt giddy. For several days afterward she had severe pain in the head, and could not see objects to her right. At the time she first noticed darkness to her right, there were a few simple hallucinations (a chair, a chicken, etc.) in the dark half-fields." Commenting on this patient, Seguin says: "A very interesting symptom not heretofore

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* Read before the Forty-Ninth Annual Meeting of the American Neurological Association, Boston, June 1, 1923.

1. Westphal: Charité Ann. **6**:350, 1879.
2. Seguin: J. Nerv. & Ment. Dis. **13**:5, 1886.

described (to my knowledge) occurred in one case. This consisted in hallucinatory images in the half field which had just become blind. The images were few and simple, such as a chicken, a chair, etc., and rapidly passed away. It seems to me that these hallucinations represented the irritation of the cortical visual center just previous to its destruction, and that they are the analogues of the localized convulsions which are now generally spoken of as indicating an irritating lesion of the cortical motor centers. . . . It is probable that further inquiry will show that hallucinations occur not infrequently at the outset of hemianopia."

Interesting as are these early reports, it was Henschen [3] who, in 1890, first definitely linked together the occurrence of visual hallucinations projected in one lateral direction, and associated with an homonymous hemianopia, with verified cerebral lesions found at the postmortem examinations. Henschen described twelve cases with visual hallucinations in which the lesion was certified either by necropsy or by operation in all but three instances. His cases were all studied exhaustively, both from the clinical and pathologic standpoint, and elaborate plates illustrating the conditions found at the various necropsies form a valuable portion of the monograph. The author related the details of the visual hallucinations with the greatest care and exactness, in each instance telling toward which side the figures appeared to the patient. It was his opinion that the hallucinations of these people were provoked by lesions irritating the occipital cortex opposite the side toward which the visual phenomena appeared. As a result of the pathologic findings he concluded that the causative lesions were nearly always outside the actual calcarine area, but nevertheless in the immediately neighboring occipital region.

In view of the more recent observations as to the site of lesions producing hallucinations, it is possible that Henschen's correlation of clinical and pathologic findings may be open to question in many instances. He seemed to have been impressed with the supposition that hallucinations of this sort must necessarily be projected toward the opposite side from that in which the brain lesion was situated. For this reason, as the majority of his cases had multiple lesions, he often assumed that a rather insignificant damage in an area which he believed should provoke hallucinations was the pathologic source of the phenomena, despite the fact that there may have been a far larger and more obvious involvement of the visual pathway in another location. Of his nine authenticated cases, three showed lesions limited to the cerebral hemisphere contralateral to the projected hallucinations (Cases

3. Henschen: Pathologie des Gehirns, Upsala: Almquist and Wiksells, I, II, III, IV, 1890, 1896, 1903, 1908.

8, 21 and 22). In these three the visual pathway was involved at many points in each case, including occipital lobes, optic radiation and thalamus, and in addition there were lesions of cuneus, lenticular and caudate nuclei and internal capsule, so it cannot be said with certainty which was the offending area. In two other cases (Cases 10 and 11) there were large infiltrating gliomas, in each instance involving the parietal, temporal and occipital lobes of one hemisphere, but Henschen attributed the hallucinations which were toward the same side as these lesions, to minute "pial hemorrhages" on the surfaces of the occipital lobes of the opposite hemispheres. In another case (Case 19) there was a tumor of the right postcentral area involving the occipital lobe, but hallucinations toward this side were attributed to an extremely small cyst and scar in the left occipital lobe. The remaining three cases (Cases 16, 18 and 28) showed multiple hemorrhages and softenings of both hemispheres so that the author's inferences as to the causative lesions must be considered as speculative. One fact, however, was clear, namely that in all instances there was a demonstrable damage to some part of the visual pathway between the midbrain and the calcarine cortex.

In addition to the exhaustive work of Henschen, interest in the localization of visual hallucinations in the occipital lobes accrued through the writings of Reinhard [4] in 1886, who published three cases with necropsies, which showed softening in the occipital areas, but the author did not mention whether the hallucinations appeared toward one side or the other. Further single reports are scattered throughout the literature during the next twenty-five years. In 1890, Dejerine, Sollier and Auscher [5] reported two cases of softening in the occipital lobes in patients who had experienced visual hallucinations consisting in definite figures of people. Also, in 1890, Wollenberg [6] reported that a patient in whom he found postmortem a tumor of the right occipital lobe, had during life a left hemianopia and hallucinations of vision. The latter were not described. In 1906, Oppenheim and Krause [7] recorded visual hallucinations in the right hemianopic field of a patient from whom a left occipital endothelioma was subsequently removed, but gave no details of the images that were seen.

The association of similar phenomena in connection with tumors or other lesions of the temporal lobes has been alluded to only in rare instances. In 1889, however, Hughlings Jackson [8] noted the occurrence of distinct visual hallucinations in a patient who was later shown to

4. Reinhard: Arch. f. Psychiat. **18**:240, 1887.
5. Dejerine, Sollier and Auscher: Arch. de Physiol. **2**:177, 184 (Jan.) 1890.
6. Wollenberg: Arch. f. Psychiat. **21**:778, 1890.
7. Oppenheim and Krause: Berl. klin. Wchnschr. **43**:1616, 1906.
8. Jackson: Brain **12**:358, 1889-1890.

have harbored a tumor in her right temporal lobe. The phenomenon was considered to be a part of her "intellectual" aura in what Jackson termed "a particular variety of epilepsy," a condition associated with the "dreamy states" which he first described in 1886 in connection with temporal lobe tumors. Later Beevor [9] in commenting on Jackson's early cases says that "these visual pictures seem to be associated more with irritation of the higher mental centers, than with that of the visual centers."

In his treatise on intracranial tumors in Nelson's System in 1920, Foster Kennedy [10] gave details of a case similar to that of Jackson. This patient's hallucination was of a woman dressed in blue who appeared on her left side. Her tumor was in the right temporal lobe, and she had a left homonymous hemianopia, the figure thus appearing in her blind field. Kennedy also reported another case of this nature,[11] in 1911, in one of a series of nine verified temporal lobe tumors, although the side on which the figure was seen was not mentioned.

Jolly [12] in 1902, and Pick [13] in 1904, thought that visual hallucinations were due to irritation of the optic tracts rather than that they were cortical, but their ideas, although plausible, were not based on necropsy or operative findings. Pick, however, cited three cases of great interest, all of which had undoubted organic lesions, in all probability involving the optic tracts rather than the occipital lobes. In this paper, referring to the work of Jolly, Pick said in part: "My aim, however, is to prove that more complicated phenomena than elementary light sensations as characterized by the scintillating scotoma—i. e., real hallucinations—may be produced in the hemianopic field of vision by localized focal or functional affections in the optic tracts. The commonly accepted idea, therefore, that such hallucinations are of value for the localization of the lesion in the occipital lobe, is not correct." The three cases which Pick reported were as follows:

CASE 1.—A man of 62 had two apoplectic seizures, the second one two weeks after the first. After the second he noted that the right half of his visual field in both eyes had disappeared, and was represented by a black shadow. He had four other slight seizures, with each a faulty understanding of what was said to him and an impulse to irrational disconnected speech. Examination showed a right homonymous hemianopia, slight right facial weakness, and slight paresis of the right arm and leg, paraphasia and paragraphia.

Hallucinations of Vision.—At one time he observed that everything on the right side of his field of vision was a reddish color, changing to green. Later he noticed for about half an hour, a dog walking along with him on his

9. Beevor: Lettsom Lecture, Lancet **1**:343, 491 and 718, 1907.
10. Kennedy: Nelson's System of Medicine, 1920.
11. Kennedy: Arch. Int. Med. **8**:317, 1911.
12. Jolly: Berl. klin. Wchnschr. **2**:42, 1902.
13. Pick: Am. J. M. Sc. **127**:82, 1904.

right side, the dog appearing on the side on which he was blind. It looked like a real dog, but the patient convinced himself that it was only a shadow. Another time a girl with a colored shawl on her head walked on his right side, but again he convinced himself that it was only a phantom.

Probable location of lesion: left gyrus angularis, and visual radiations.

Case 2.—A man of 56 received a head injury one year before examination. He had numbness of the left hand and leg, pains in the left big toe, left hemianesthesia and astereognosis, disturbance of vision toward the left, and a left homonymous hemianopia.

Hallucinations of Vision.—In the blind half field of vision he noticed all sorts of figures and imagined sometimes that some one was sitting next to him.

Probable location of lesion: posterior knee of the internal capsule, pressing on the optic thalamus and the left geniculate body at this point.

Case 3.—Similar to Case 2 with a lesion in the same place presumably.

It may be said also, that the ideas of Pick and Jolly were borne out by the report of a case by de Schweinitz [14] in 1891, in which definite figures (chairs, tables, etc.) were seen in the left field which subsequently became hemianopic; at necropsy a gummatous infiltration was found at the base pressing on the right optic tract. It also happened that in one of the cases recorded by Dejerine, Sollier and Auscher, in 1890, there were adhesions around the left temporal lobe, as well as softening of the left cuneus, in a patient who had shown a right homonymous hemianopia and had experienced visual hallucinations. Recently Cushing [15] has referred to the frequent occurrence of hallucinations of vision in temporal lobe tumors, and it is with an elaboration of the visual phenomena of these cases that this communication proposes to deal.

In the series of 873 verified cases of brain tumor from Cushing's clinic there have occurred seventy-two in one or other of the temporal lobes. Of these, it has been recorded of seventeen that there occurred visual hallucinatory phenomena of one sort or another. What we have regarded as definite hallucinations of figures appeared in twelve of these seventeen patients. The other five showed visual phenomena of a less elaborate and exact character.

The typical visual hallucination is a phenomenon that is extremely striking when elicited from a patient who has experienced the sensation. As Cushing has said in speaking of temporal lobe tumors, "it seems to be part and parcel of the uncinate seizure," and therefore apparently definitely bound up with those phenomena which have been regarded as being produced by irritation of the temporal lobes. At least, in this series, the visual phenomena were associated with uncinate attacks or "dreamy states" in thirteen of the seventeen cases. The

14. De Schweinitz: New York M. J. **53**:514, 1891.
15. Cushing: Brain **44**:341, 1921.

usual story obtained from the patient is that during the few moments of "unreality" or "dreamy state," often while experiencing the olfactory or gustatory aura, there appear to them, usually toward one side or the other, the figures or sometimes the shadows of people, animals or inanimate objects. These figures sometimes seem grotesque, sometimes fairly natural. Often they are diminutive or more rarely, enlarged. In some cases the hallucination is stationary, in others it may seem to be coming toward or going away from the patient. In one of our cases the whole phenomenon was so repulsive that the patient dreaded even telling about it. In nine instances the hallucinations have appeared either in the blind half-fields or, if no hemianopia was demonstrable, toward the side opposite the tumor, while in the others the phenomena were not recorded to have been projected toward either side. The twelve cases showing definite figure formation fall naturally into two groups from a clinical as well as a pathologic standpoint.

GROUP I. CYSTIC GLIOMAS

In the first group there are six cases, in all of which a temporal lobe gliomatous cyst was demonstrated either at operation or at necropsy. Three of these patients have remained alive and well, one for nine years, one for three years, and the other for six months since operation. The other three have died. An an example of a typical case in this group we may cite the following:

CASE 1.—*Clinical History.*—J. B., a schoolboy, aged 11, was referred to the Peter Bent Brigham Hospital on Dec. 14, 1915, by Dr. N. P. Breed of Lynn, Mass. His family and past history were unimportant. About one year before admission he began to have pain in his eyes and through his eyeballs. This pain was continuous and became very severe. Associated with it he had headaches which were largely frontal. Nausea and vomiting accompanied the headaches from the start, and he had tinnitus in the right ear at intervals during the month previous to admission. Also during the month before admission he complained of slight blurring of vision, and diplopia for one week. On two occasions he had experienced uncinate gyrus seizures with hallucinations of smell.

Hallucinations of Vision.—One day during the week before admission he described having seen the figure of a boy on the wall. "The boy had his hands behind him and was walking." This phenomenon occurred at eight o'clock in the evening, while the patient was reclining in a chair. He said it was not a dream, because he had his eyes open. The figure appeared on his left side. He also had another hallucination on the night following the first one. It occurred in the middle of the night, and he said, although the rest of the family were asleep, he himself was lying in bed with his eyes open. He saw "a man dressed in white sitting by the fire, bending over to tie his shoe." This figure also appeared on his left side. The patient was sure he was not asleep, as he closed and opened his eyes several times, and the figure was always there. While in the hospital he had still another hallucination. On this occasion he was lying in bed, late in the afternoon. The room was

rather dark. He saw six children sitting around a desk on the wall, and also saw the figure of a girl in the room. On asking the nurse to turn on the light, the figures vanished.

Neurologic Examination.—Showed the presence of bilateral choked disk with an elevation of from 3 to 4 diopters. The left pupil was slightly larger than the right, and there was a left upper quadrantal homonymous visual defect.

First Operation.—Dec. 31, 1915, Dr. Cushing performed a combined exploration and decompression of the right temporal region, by turning down a bone flap in this area. The lower temporal convolutions were somewhat flattened and soft. A needle was introduced into the second temporal gyrus and at a depth of 4 cm. a gliomatous cyst was encountered from which a few cubic centimeters of typical yellow, easily clotting fluid were evacuated. The bone flap was then replaced.

Convalescence was uneventful. Jan. 20, 1916, he had another hallucination. This time he saw the figure of a lady who appeared on his left side. She was "dressed and had her hat on."

All of the patient's pre-operative discomforts were relieved, and the choked disks receded to 1.5 diopters on each side. He was discharged Jan. 22, 1916.

The symptoms, however, recurred within a short time and he was readmitted Feb. 14, 1916. The decompression area was bulging, and there was a beginning weakness of the left face, arm and leg, with increased reflexes on this side. He now had a complete left homonymous hemianopia.

Second Operation.—Feb. 22, 1916, Dr. Cushing again exposed the right temporal region and partially removed the soft cystic glioma which was disclosed. He was discharged March 12, but failed rapidly and died April 11, 1916. No necropsy was obtained.

The five other cases in the series are almost exact counterparts of the example just cited in both their neurologic and pathologic aspects. The only case in this group which came to necropsy may be cited fully.

CASE 2.—*Clinical History.*—C. J. M., an Italian laborer, aged 31, was referred to the hospital Jan. 30, 1914, by Dr. J. W. Courtney of Boston, Mass. The family and past history were unimportant, except for the fact that he had fallen out of a tree when a boy and struck the back of his head. This was apparently not a serious injury and he was up and about feeling very well the next day. He had been in good health until three or four months before admission, when he began to get "run down," nervous, and had a general feeling of weakness. Five weeks before coming to the hospital he began to have headaches, vomiting, and dizziness. At this time his vision commenced to fail, he became much confused mentally, and staggered drunkenly in walking. He complained also of buzzing in his right ear, and of impaired hearing in this ear. He also described some hallucinations of smell which he had experienced a few weeks before admission.

Hallucinations of vision.—Feb. 4, 1914, when the nurse was about to take his temperature, he exclaimed that he had "just seen a bird in the room, flying about a yard or two in front of him. It was a very beautiful bird," and he reached out his hand several times to grasp it. It remained fairly stationary for many seconds in front, and slightly to his left side.

Neurologic Examination.—Examination revealed bilateral, high grade choked disks of 6 diopters, left homonymous hemianopia, slight ataxia and dysmetria of the upper and lower extremities, with unsteadiness in standing and walking.

Operation.—Feb. 6, 1914, Dr. Cushing explored the right temporal region by a bone-flap reflection. A gliomatous cyst of the right temporal lobe was evacuated, and a bony defect was left as a decompression.

He had a stormy convalescence, and gradually declined after the operation. death occurring May 22, 1914.

Necropsy Findings.—The brain was removed after formalin fixation by injection through the carotids. Transverse sections were made beginning at the tips of the frontal lobes and proceeding posteriorly. These sections showed a tumor mass occupying a large portion of the right temporal lobe. The superficial cystic portion of the growth was collapsed, and from this area it extended inward even beyond the median line, involving the optic thalamus, and occluding the right lateral ventricle. Posteriorly, the tumor occupied the more medial portion of the temporal lobe and finally ran downward into the aqueduct of Sylvius. There was no involvement of the occipital lobe whatever. Microscopic sections showed a typical glioma.

The other four cases which come into this group of gliomatous cysts need not be given in detail. It is interesting, however, to record the hallucinations, as further examples of the definite figure formations under discussion. In every instance the lesion was demonstrated at operation to be situated in the temporal lobe, involving the optic radiation certainly in three instances as shown by a contralateral hemianopia. In the other case no fields were taken. The description of the phenomena which appears in the history of the first of these is as follows:

CASE 3.--H. L., during one night constantly saw a woman friend in the room, and wanted her husband called to talk to this woman. The figure stayed in the room all night, and moved about but did not talk. It was always on the patient's left side (she had a left homonymous hemianopia and a right temporal lobe cyst). When the patient turned her head to follow the figure, it would also go to the left and disappear.

On another occasion, while being examined, she saw a man with a gold helmet on his head sitting in a chair beside the examiner. He did not leave the room until the examiner left. She could not recognize the figure's face, nor describe it clearly. Both the examiner and the figure were to the left of the patient.

This woman died after the cyst had refilled and been tapped several times.

CASE 4.—A. C., the record of the second patient is rather more brief, but no less definite as to the figures seen. He also had a cyst of the left temporal lobe, and was alive and well at our last report, three years after a simple evacuation of the cyst contents. This patient had a period of three days in the course of his illness during which he felt dazed all the time as if having one of his petit mal seizures. At this time he complained constantly of seeing horses, cows, pigs and ship-wrecked men, but there is no mention as to the side on which they were seen.

CASE 5.—E. A. J., a woman of 43, who had several evacuations of a right temporal lobe cyst. Her visual hallucinations consisted in seeing "colored lights and processions of queer figures marching on the ceiling." She has remained well for nine years since the first operation.

CASE 6.—G. L. R., a woman of 50, who was operated on Dec. 2, 1922. Her visual hallucinations had been present for one year before admission, and always accompanied typical uncinate gyrus attacks, which she might have once in two or three weeks or as often as five or six times a day. At these times she said, "I see figures approaching me from the left side. They are never very distinct, but sometimes seem like little people, a little old woman, etc." This patient had a left homonymous hemianopia, and at the operation a small deep gliomatous cyst of the right temporal lobe was tapped.

GROUP II. SOLID GLIOMAS

The next group, also showing definite figure phenomena, is that of the solid gliomas, of which there were six in the series. These cases from the standpoint of treatment are exceedingly unfavorable in comparison with the foregoing class, and all have sooner or later succumbed to the effects of the lesions. Chief interest here centers around two patients who were followed in the hospital over long periods of time, and whose hallucinations have been recorded in detail.

CASE 7.—*Clinical History.*—E. G., a woman of 28, was referred to the hospital June 28, 1913, by Dr. Henry Boynton of Townsend Centre, Mass. Her family and past history were unimportant, save that her mother had once been in an insane asylum for six months.

Six months before admission she commenced to have headaches which were at first suboccipital, radiating to the vault, but later largely frontal in location. Vomiting was associated with the headaches and tended to be projectile. Her vision began to fail at the time of the onset of the headaches, at first merely a blurring of vision, but later the decline in eyesight was very rapid. Double vision and dizziness were present two to three months after the onset of symptoms, and she also had at times a numbness and prickling sensation in her right arm and leg. Subjectively there was a complete loss of the sense of smell, and she had also noted some disturbance in taste for two or three months previous to her entrance into the hospital.

Physical Examination.—On admission she showed some muscular weakness of the right hand as compared with the left, right abducens palsy, left pupil slightly larger than the right, absence of taste on the right side of the tongue (anterior two-thirds), and bilateral choked disks of from 6 to 7 diopters, with vision reduced to 20/200 O.U.

Operation.—July 8, 1913, Dr. Cushing performed a right subtemporal decompression. The brain was under extremely great tension, but no surface tumor was disclosed. The patient made a good operative recovery; the choked disks subsided to 3 diopters; the headaches, vomiting and dizziness disappeared, and she partially regained her olfactory sense. She was discharged July 27, 1913.

Course.—She was readmitted for short periods of observation Oct. 4, 1913, and June 25, 1914. On the latter date headaches and vomiting had recurred, and vision was entirely gone. Her final admission was Aug. 11, 1914, and she remained in the hospital until her death May 31, 1915. During this time she was under constant observation, comfortable for the most part, although she commenced having generalized convulsions, and it was in one of these that she died.

This patient's voluminous record contains many detailed accounts of visual hallucinatory phenomena from which the following are quotations.

Hallucinations of Vision.—Nov. 30, 1914. During the past month the patient has at intervals sat up in bed and turned rapidly to the right as if following something with her eyes. On questioning it was found that on one occasion she continually pointed as she turned and asked the nurse if she "did not see the leopard on the wall?" She saw other animals at different times, and occasionally people, always toward the right.

Feb. 20, 1915. Yesterday and today she again had hallucinations appearing on her right side. She sat up in bed and looked far around to the right. When asked what she saw she said, "Little people, and they are yellow."

Feb. 22, 1915. On questioning today she said there is "a steady glow of light over to the right side." Later she looked to the right and said, "Yes, I see a red colt there now. It is moving slowly and is not very big."

March 28, 1915. She has constantly seen the "red lights" to her right. Today she "saw a dog standing out in the field. He had pointed ears and brown spots. He moved his head from side to side and seemed to be waiting."

May 23, 1915. She has frequently seen red objects, "like children's balloons" floating to her right. This morning they were directly ahead and one was green. She said that once or twice the red objects had been to her left. Yesterday she said "there were a lot of kittens playing over there" (pointing to her right).

May 31, 1915. Death occurred following a convulsion.

Pathologic Report.—The brain was removed after formalin fixation by injection through the carotids. Transverse sections were made beginning at the tips of the frontal lobes and proceeding posteriorly through the occipital. These sections showed a diffuse glioma occupying the more medial portion of the right temporal lobe. It extended inward to the basal ganglions, involving the optic thalamus and occluding the right ventricle. It also spread outward across the visual pathway, nearly to the lateral surface. There was no involvement of the occipital lobe by the tumor. Microscopic studies confirmed the gross diagnosis of glioma.

Comment.—It is unfortunate that this woman was so nearly blind at the time she came under observation because no very accurate record of her visual fields could be obtained; there is little doubt but that she must have passed through the stage of a left homonymous hemianopia previous to total loss of eyesight. At all events, the visual hallucinations were projected toward the right side, and the tumor was in the corresponding temporal lobe. In addition to temporal lobe, however, other neighboring structures were evidently involved, such as optic thalamus and septum lucidum. The right visual pathway in the temporal lobe was seen to be distinctly infiltrated. Obviously then, one cannot associate the hallucinatory phenomena with any one separate structure, except to eliminate other portions of the brain than the temporal lobe and its immediately contiguous area.

The second patient in this group was one of the older cases in Dr. Cushing's Baltimore series, and was observed at intervals over a period of seven months.

Case 8.—*Clinical History.*—J. K., a schoolboy 16 years of age, was referred to the Johns Hopkins Hospital Sept. 21, 1908. His family and past history were unimportant. During the two years previous to admission he had not been as bright and active as formerly. For one year he had been having focal attacks without loss of consciousness, involving his right side, and associated

with olfactory and gustatory phenomena. For two years he had had headaches and many vomiting attacks and during the ten months previous to his entrance to the hospital vision had been failing.

Visual Hallucinations.—On many occasions he said that he saw distinctly "a crowd of men playing cards and having a good time." They were usually acquaintances of his playing euchre, and the game inevitably ended in an altercation among the participants. This visual picture was always the same, and was part of the aura of his petit mal seizures in which there were nearly always uncinate gyrus features. He never described any other scene than the one mentioned, nor any other figures.

Neurologic Examination.—This revealed a slight right facial weakness, and tenderness over the left temporal region. He had bilateral choked disks of 5 diopters. Otherwise, examination was entirely negative.

Operation.—Sept. 23, 1909, Dr. Cushing turned down a bone flap over the left temporoparietal region. There was general intracranial tension, but no lesion was disclosed. A subtemporal decompression opening was left and the wound was closed. His convalescence from this procedure was uneventful; the choked disks subsided and he was discharged Nov. 3, 1908. Nov. 23, 1908, he was again admitted, having declined markedly in the interim. December 5, the right hemisphere was explored without encountering a lesion, and a subtemporal defect was left on this side also. He was discharged Jan. 7, 1909, and readmitted February 10. At this time his memory had begun to fail, and he had a beginning aphasia. His sense of smell was greatly affected. On this admission it was determined that he had a right upper quadrantal homonymous field defect.

Feb. 13, 1909, his left temporal lobe was again explored and the cortex incised, but no tumor was demonstrated. He was discharged March 9, 1909, and died April 7.

Pathologic Report.—The brain was removed after fixation by injection through the carotids. Serial, gross, transverse sections were made of the entire brain, which showed the growth to be confined to the left temporal lobe. A section through the midtemporal region showed the largest extent of the tumor; it occupied the medial portion of the lobe, and at this level was about the size of a hen's egg. The optic radiation was evidently involved and the growth had also infiltrated the optic thalamus on this side. Another section, from 2 to 3 cm. farther back, showed the posterior tip of the tumor, in the uncinate region. There was no involvement of the occipital lobe. Microscopic studies showed the tumor to be a glioma.

The other four cases in Group II need not be detailed. All had definite figure hallucinations; in one these were projected to the side opposite the brain lesion, but in the others it was not stated on which side the images were seen. Three of the tumors were demonstrated at operation, and the other at necropsy.

GROUP III. CASES WITH LESS DEFINITE HALLUCINATIONS

The five remaining cases of temporal lobe tumors showing less well defined visual hallucinatory phenomena may be placed in a group by themselves. The character of the hallucinations was either not accurately described in the records, or they were not the clear cut figure

phenomena such as were observed in the foregoing descriptions. In the history of one of these patients it was simply mentioned that he had visual hallucinations, and at operation a gliomatous cyst of the right temporal lobe was disclosed. In another patient, from whom an encapsulated glioma of the right temporal lobe was removed, it was noted that "he had hallucinations of seeing people and hearing them speak to him."

The other three cases deserve more than passing mention. One of these patients had for two months before admission the hallucination of "seeing something like a big red wall on her left side." She said it was constantly on that side and had never moved to any other position. Her tumor was not localized before operation, and owing to the fact that she was left handed, a left decompression was performed instead of the usual right sided procedure; no lesion was found. She lived for three months, and at necropsy a huge encapsulated tumor occupying the right temporal lobe was demonstrated. This growth macroscopically was taken to be an endothelioma, but microscopic studies proved it to be a mixed glioma and neuroblastoma.

Concerning the remaining two patients there was one observation which is of special interest. One patient was a physician who suffered from uncinate gyrus seizures which were inaugurated by his thinking there was something to be seen on his right side, but when he looked toward the right there was nothing. The striking thing about his visual phenomena, however, was the fact that when having a petit mal seizure all his special senses seemed exaggerated. So far as vision was concerned, this manifested itself by making objects appear abnormally large to him, a condition noted by Josefson [16] in 1913, in recording the visual hallucinations of a patient who had an occipital lobe glioma. Josefson gave the name macropsia to this phenomenon. In addition to the magnified size of objects, our patient also stated that colored objects, such as flowers, also looked "extraordinarily beautiful" to him, a condition which might be given the term "kalopsia." This man had a right homonymous upper quadrantal field defect and at operation a cystic glioma of the left temporal lobe was disclosed. He lived ten months after the evacuation of the cyst. The other patient also said that he often saw in his blind half fields, colors which seemed uncommonly beautiful.

SUMMARY

From a survey of a large number of the publications which have dealt with hallucinations of vision in patients who have suffered from organic disease of the brain, it would seem that, essentially, three views have been held as to the brain area provoking this phenomenon.

16. Josefson: Deutsch. Ztschr. f. Nervenheilk. **49**:341, 1913.

By far the largest number of writers, among whom Henschen, from his work, stands out preeminently, have regarded the occipital lobes as the offending region. A careful review of Henschen's cases and those of others who hold his views has revealed the fact that the lesions depicted have been too diffuse and widespread to admit of a definite localization of their visual hallucinations in the occipital lobes alone. Further evidence against the occipital lobes as the source of figure hallucinations is had from the cases of occipital lobe tumors in the series at the Peter Bent Brigham Hospital. Of these there have been eleven, and in none were there recorded phenomena which could be interpreted as imaginary visual images such as were present in the patients with temporal lobe lesions. Likewise, from the temporal cases just reported there is the negative evidence of the fact that, in those which came to necropsy, none showed an extension of growth to the occipital lobes.

The second theory, advanced by Pick and Jolly, that this phenomenon was provoked by lesions purely of the optic tracts, has no basis authenticated by necropsy findings so far as their cases were concerned, although borne out by one or two instances reported by others.

The third view is that brought forward by Hughlings Jackson, namely, that visual hallucinations, although occurring in certified tumors of the temporal lobe, were believed to have been provoked by irritation of the higher mental centers, and therefore represented a distant effect of the growth, rather than a local pressure or irritation phenomenon. Regarding the temporal lobe origin of visual hallucinations we are in full accord with Jackson, but from the evidence in our series it would seem that the phenomenon is called forth by a direct irritation or pressure effect in the temporal lobe itself, as the visual pictures are so often precipitated in association with, and as a part of the olfactory and gustatory aura of, a typical uncinate seizure.[17]

In the series here presented there have occurred visual hallucinations in seventeen patients with tumors which have been shown to have occupied at least some portion of one temporal lobe. In six of these seventeen cases the exact extent of the lesion could be demon-

17. This interpretation is apparently in accord with that of Kennedy expressed in his discussion of Cushing's paper on temporal lobe tumors in the Transactions of the American Neurological Association, 1921, p. 420. In this discussion Kennedy also elaborated his theory as to why complex figure hallucinations should be provoked by irritation of the temporosphenoidal region. This theory is based on the idea that in very early life the temporosphenoidal lobes act as a storehouse for infantile memories, a function which is later taken over by other brain areas. Thus, in the early days "memory pictures may be laid down and pass into unconsciousness, only to be called back to consciousness in the presence of gross irritation of this area."

strated by postmortem findings, and in each instance there was seen to be extensive involvement of the temporal lobe, together with that portion of the visual pathway passing through it. Other structures in the immediate neighborhood were likewise encroached on, most noteworthy among which was the optic thalamus. In none of these cases was there the slightest involvement of the occipital lobe.

The other eleven cases all had temporal lobe tumors or cysts demonstrated at operation. The size and extent of these lesions could only be estimated by what was actually visible, or by other evidence of involvement elicited by neurologic examination. Certainly in Group I, Cases 1 and 3 must have had very large tumors, as they both had a complete hemianopia, and although one of them lived for a year after operation, it was only because the cystic portion of the growth could be tapped from time to time. Cases 4, 5 and 6 of this group were alive and well at our last reports. Two of them certainly had relatively small cysts, and in Case 5 the fields never advanced beyond the stage of an homonymous quadrantal defect. In Case 4 no fields were taken.

In Group II, of the cases not detailed, one had tumor which was first disclosed 3 cm. below the cortex. It was evidently a slowly growing lesion for she did very well for four years after a decompression. At the end of that time there was great protrusion of the decompressed temporal area, and at a subsequent operation when a large amount of tumor was scooped out, there was no evidence that the growth extended posteriorly. The other three cases also had large gliomas which could only be partially extirpated.

In Group III, of the three cases not coming to necropsy, one had a small encapsulated glioma and the other two had gliomatous cysts with probably extensive solid growth as well.

CONCLUSION

The evidence from this series of cases would seem to show that a highly complex variety of visual hallucinations may be caused by tumors of the temporal lobe. In every instance in which it could be ascertained, the visual pathway was involved by the lesion, and in the six cases coming to necropsy there was involvement also of the optic thalamus. In eight instances out of the fourteen in which note was made of the laterality of the phenomenon the hallucinations were projected toward the side opposite the lesion in the brain.

DISCUSSION

DR. MORTON PRINCE, Boston: Clinically, a symptom may have localizing value when correlated with a given focal lesion. Thus, hallucinations might frequently be correlated with lesions of the temporal or occipital lobe. On the other hand, from a psychopathologic point of view, it would not justify the inference that hallucinations are a function of either one of those lobes.

The hallucination may, and in fact undoubtedly must be, the product of conscious processes correlated with the brain as a whole, or at least with many and various distant areas. The mechanism is to be sought in either release of inhibition, permitting denervated mental symptoms to function autonomously and automatically, or in irritative impulses sent to distant regions and instigating similar complex mental processes. A hallucination is very complex psychologically, involving perceptual images, cognition and various other processes. It is also automatic, and probably the expression of subconscious processes. It is incredible that such a complex phenomenon should be correlated with any particular focus in the brain.

Dr. M. Allen Starr, New York: This paper states that visual hallucinations occurred in but thirteen of Dr. Cushing's 850 cases, and that in each case the tumor was located in the temporal lobe. Therefore, it seems to me that the visual hallucination is positive evidence, a localizing symptom of a lesion in this particular region, and Dr. Morton Prince's objection is not well taken. If on the contrary, his position is well taken, we should have a history of visual hallucinations from tumors everywhere in the brain.

Dr. Harvey Cushing, Boston: There is a patient in the wards that might be of interest in this connection. She has homonymous hemianopia from an occipital lobe glioma. Her history records that she has been having visual hallucinations of a crude sort, such as seing a crescent of light. This is not the sort of hallucination we associate with tumors of the temporal lobe, a visual impression associated in some ways with Hughlings Jackson's dreamy states. Her subjective images are very crude, and are not particularly vivid images.

Dr. Charles K. Mills, Philadelphia: I have reported cases with hallucinations due to lesions of the temporal lobe. The work recorded by Dr. Horrax indicates the type of work that should be done with regard to determining the origin and evolution of hallucinations, rather than by the older method of looking at crystals. I believe that the temporal lobe is the most likely situation of irritative lesions causing hallucinations. It must always be remembered, however, when we speak of temporal and occipital that the real region morphologically is temporo-occipital.

Dr. Prince: There is one clinical aspect which deals with localization, and another which concerns the psychopathology. I think, as a phenomenon, a hallucination may well be a localizing symptom; that is a clinical matter—a question of correlation. Meynert attempted to explain hallucinations by assuming an area which, when destroyed, released inhibition. Thus a lesion of the temporal lobes or elsewhere, releasing inhibition, permitted hallucinations to be created elsewhere. In this way hallucinations may be localizing symptoms, and, I believe, according to the evidence that we have heard presented here, they probably are. It is another question whether the hallucination itself is localized there. That is a psychopathologic question and does not at all negative the fact of the localizing value of the symptom. I do not think it is possible, basing this statement on such knowledge as we have, that irritation of any particular focal area can produce such a complicated psychologic phenomenon as a hallucination, comparable as some are to a cinema picture.

Dr. S. A. Kinnier Wilson, London: I believe that distinction should be made between the crude and the highly organized type of hallucination. From the clinical point of view, I think that the crude type is more likely to be

associated with lesions in the receptive areas of the occipital or temporal lobes, whereas the more highly organized types are more likely to be associated with lesions which have their origin in the cortical association areas. It is of interest to note whether there is an element of familiarity in the phenomenon, whether it occurs in the same form each time, or whether the patient feels it is different from anything he has experienced before. There is another form in which the patient has a "visual memory aura," when the hallucinations appear like a panorama, going deep down into old incidents in his life, into long-forgotten events, which appear again in the form of hallucinations. Though I recognize the force of Dr. Prince's contention, as a clinician I associate the elaborate type of visual phenomenon with temporal or temporo-occipital lesions, that is lesions in association fields, and the cruder type with lesions of the receptive zones.

Dr. Harvey Cushing, Boston: In connection with what Dr. Wilson has said concerning the hallucinations of a more vivid type, I would like to recall an early case of my tumor series in which these impressions were fully recorded. The patient was a small boy whom I had had under observation for a long time, and who ultimately died from a temporal lobe tumor. I saw him in many of these attacks. They were always inaugurated with gustatory and olfactory impressions, and ended with a vivid hallucination. The attack would begin with a vague stare. He might be in the midst of a conversation, when he would stand, become salivated, go through chewing movements, and finally turn and look to one side. This was followed by a horrified expression. The whole seizure lasted possibly thirty seconds. He always saw the same scene —his father playing cards with a group of men, and getting into a quarrel with them; the game always ended in a squabble in which these people were fighting. (As a matter of fact, his father did not play cards at all.) He never became accustomed to this scene and was always equally shocked and horrified by it. It was a very vivid hallucination associated with what we would call a typical dreamy state. I never saw the patient have a convulsion.

Dr. Horrax, in closing: As to the mental attitude of the patients toward these phenomena, it has been recorded in some instances in our histories, and in cases reported elsewhere, that the patients were quite aware that these were hallucinations. In other words, they would see the figures off to one side and convince themselves they were not real by feeling or looking around, or seeing that there was not an actual object beside them. No cases of brain tumor, except those in the temporal or temporo-occipital regions, in Dr. Cushing's series had hallucinations of this sort. There might have been vague hallucinations, such as are present in other diseases, or in inorganic troubles, but none of these definite figure hallucinations of the type we have recorded. In regard to Dr. Kinnier Wilson's question, that is the distinction we wished to draw; the crude sensations of scintillating scotoma or the crude sensations of light are associated definitely with occipital lobe tumors. In the patient we exhibited this morning, the definite diagnosis of occipital lobe tumor could be made from her crude hallucinations, whereas the more elaborate hallucinations we believe to be associated with temporal lobe tumors. The cases which we reported are all temporal lobe tumors, five or six of which have been confirmed by necropsy.

GASTRIC SECRETORY FUNCTIONS IN THE PSYCHOSES *

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In works on psychiatry, gastric and other secretions are generally assumed to be diminished in depressed or melancholic states, and increased (less clearly) in excited or manic states.[1,2] Less definite statements are made in regard to dementia praecox.[3] In many depressed patients, moreover, there are definite gastro-intestinal symptoms and curious somatic delusions which are well exemplified in some of our cases. They were aptly described by Clouston.[4] Independently of psychiatry the observations of physicians and physiologists [5,6] point to a marked influence of the mental, and particularly of the emotional, state on gastro-intestinal functions, both secretory and motor. While this supposed correlation between the mental and bodily functions seems to be reasonable, and clinically well substantiated, we have been able to find no studies by modern methods (fractional test meals and roentgenologic observations) confirmatory of these assumptions. The present study was undertaken to fill this gap in our knowledge. Our observations are as yet obviously too few to be decisive even in respect to secretion. We intend to continue them and to include motility observations by Haudek's roentgenographic method.

Our patients were all in the psychiatric wards of the Pennsylvania Hospital. The complete fasting contents were removed in the morning and an Ewald test breakfast was usually given with the tube still in place. We used as a rule small rubber tubes (No.12) weighted at the

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* From the laboratory of the Pennsylvania Hospital, Department for Mental and Nervous Diseases.

* Read before the American Gastro-Enterological Association, May 1, 1923, and the Philadelphia Psychiatric Society, May 11, 1923.

1. Stransky, E.: Das manisch-depressive Irresein, Leipzig: 1911.

2. Kraepelin, Emil: Manic-Depressive Insanity, Chicago: Chicago Medical Book Co., 1921, p. 48.

3. Kraepelin, Emil: Dementia Praecox, Chicago: Chicago Medical Book Co., 1919, p. 86.

4. Clouston, T. S.: Lectures on Mental Diseases, Philadelphia: 1884, p. 73.

5. Miller, Bergeim, Rehfuss, and Hawk: Psychic Secretion of Gastric Juice in Normal Men, Am. J. Physiol. **52**:1 (May) 1920.

6. Cannon, W. B.: The Mechanical Factors of Digestion, London, 1911, p. 210; Bodily Changes in Pain, Hunger, Fear and Rage, New York: 1915.

tip by a perforated lead shot and supplied with lateral perforations. These tubes could be used equally well through the nose or mouth. Fractions were removed at fifteen minute intervals by the Rehfuss method. When the stomach was apparently empty, 100 c.c. of water was injected and withdrawn to demonstrate complete evacuation. The total and free acid of the fasting contents and of the fractional specimen of gastric chyme were estimated in the usual manner. A specimen removed in from sixty to seventy minutes after the beginning of the meal was recorded as the "one hour" specimen for comparison with standard "Ewald" figures. Pepsin was tested in this specimen by the Mette method. In most cases quantitative estimations of lipase and trypsin were made in the fasting contents and in each of the gastric fractions. Gaultier's methods as modified by one of us [7] were employed. Regurgitation of bile and the time of complete evacuation were recorded.

In all patients routine examinations were made of blood, urine, Wassermann reaction and blood pressure; and in many, spinal fluid findings, blood cultures, blood chemistry and other data were secured. In twenty-four of the forty-six cases the basal metabolism was estimated by the Roth-Benedict apparatus. In most cases this was within normal limits. In four cases there was definite increase or decrease of the basal metabolism, but a careful comparison with the gastric findings failed to show any correlation. These tests have incidentally proved valuable as a measure of the patient's cooperation. Thus in Group *A* all were sufficiently cooperative. In Groups *B* and *C* (clinically the most depressed, and those showing the most marked changes in gastric secretion) it was possible to take the basal metabolism in only four of twelve cases. In Groups *D* and *E*, which appeared casually similar (depressed and anxious), only one of the twelve cases lacked the necessary cooperation; another remained untested for other reasons.

We tabulated all the important gastric findings in our series of cases and then arranged them under important clinical headings—manic-depressive psychosis (including involutional melancholia), psychasthenia, dementia praecox, and miscellaneous psychoses due to somatic disease. Subsequently we arranged these cases under each disease group in the order of the gastric acidity (as determined at one hour). In doing this we had in mind Carlson's dictum [8] that "pathological deviation in acidity is always in the direction toward anacidity." When we had finished we were struck by the close correspondence between the degree of emotional depression and lack of cooperation on one hand,

7. Lueders, C. W., and Bergeim: Quantitative Determination of Trypsin and Lipase in Gastric Contents, Am. J. Physiol. **66**:297, 1923.

8. Carlson, A. J.: Some Practical Aspects of the Physiology of Digestion, Tr. Am. Gastro-Enterol. A., 1919, p. 232.

and the reduction in acidity or delay of secretory response on the other. In the following pages is an analysis of the table with brief clinical histories (including positive data only).

SUMMARY OF CASES IN GROUP A

The group contains four cases, predominantly manic. The emotional tone varied from elated to normal. The secretory response was: high acidity (hyperacidity); normal or continuous curves; evacuation as a rule normal, occasionally slightly delayed.

The findings are not inconsistent with the emotional state, that is, the motor and secretory functions are normal or increased. There is no evidence of organic disease of the stomach. There were unexplained blood and leukocytes in the fasting contents of one case.

REPORT OF CASES

CASE 1. *Manic-Depressive Psychosis, Hypomanic Type.*—Ada B., a woman, aged 21, was admitted Dec. 30, 1922. The onset of the present attack had occurred two months before admission. No gastric symptoms were present or had existed previously; the bowels were negative. General and laboratory examinations were negative.

There were: slightly increased gastric acidity; normal curve with a tendency to the continuous type; slightly delayed evacuation time. Earlier test meals showing abnormal findings were explained by gagging and duodenal regurgitation. The findings suggest slightly exaggerated secretory response, and are not inconsistent with a hypomanic state.

CASE 2. *Manic-Depressive Psychosis, Manic Type.*—Anna N., a woman, aged 71, who was admitted Sept. 21, 1914, had had twenty-six distinct periods of excitement. (Subsequently, she died during a period of depression. At necropsy, extensive arteriocapillary fibrosis was found). At the time of the examinations she was hypomanic, but she cooperated. Appetite was good and nutrition excellent, though she was slightly obese. There were no gastrointestinal symptoms; the bowels were regular. There were no abnormal physical findings. The pulse rate was 90; blood pressure, 186 systolic and 94 diastolic.

The gastric findings were: normal or moderately increased acidity; normal curve; prompt evacuation. The findings suggest slightly exaggerated secretory response and are not inconsistent with the hypomanic state.

CASE 3. *Manic-Depressive Psychosis, Circular Type.*—Alice B., a woman, aged 47, was admitted July 18, 1917. The disease began at 39 with depressions followed by normal intervals, and later manic phases. Recently there had been twenty-eight days of severe mania, during which she was exhilarated, noisy, unreasonable, untidy, etc.; this was followed by one day of normality and then twenty-eight days of mild or moderate depression. Tests were taken during the mild depression. The patient was constipated during the depressed periods and had hemorrhoids. Polyphagia occurred during excitement. She ate poorly in the depressed period. She was sallow and weighed 109 pounds (50 kg.), pulse rate, 76; blood pressure, 186 systolic, 118 diastolic during the depression, and 210 systolic, 150 diastolic in the manic stage. The urine showed occasionally a faint trace of albumin and a few hyalin casts (it had been negative recently).

The gastric findings were: hyperchlorhydria with a tendency to a continuous curve; slight delay in evacuation; no duodenal regurgitation. The findings suggest slightly exaggerated secretory response with possibly increased tone of the pylorus; they have no relation to the psychic state.

CASE 4. *Manic-Depressive Psychosis, Manic Type.*—Alma P., a woman, aged 41, was admitted June 4, 1922. The present attack began in June, 1922, following the illness and death of her husband. There were brief periods of mild depression but usually she was exalted. At the time of the tests she was hypomanic, but cooperative. Appetite was good and there were no gastro-intestinal symptoms. Nutrition was good, the weight being 129 pounds (58.7 kg.), and the color was good. General and laboratory examinations were negative.

The gastric findings were: moderate hyperchlorhydria, with a normal curve tending to a continuous type; prompt evacuation. The findings suggest slightly exaggerated secretory response, and are not incompatible with the emotional state.

SUMMARY OF CASES IN GROUPS B AND C

In the twelve patients in these groups there is apparent agreement between the depressed emotional state and the altered secretory findings (hypochlorhydria or achlorhydria). Other factors, particularly malnutrition, were possibly operative. The findings in Group *B* are more striking than those in Group *C*. Most of these patients were resistive or did not cooperate.

Group B.—Six cases. All were markedly depressed and either anxious or resistive. Five of the patients were more or less emaciated, and several had distinct dyspeptic symptoms, recurrent diarrhea, and other gastro-intestinal disorders. Four of the patients showed achlorhydria throughout (two, in fact, achylia). The other two showed marked hypochlorhydria of the delayed type. All had low or absent pepsin; duodenal ferments were reduced in the majority. Motor function was normal in all; early emptying occurred in four or five. There was evidence of pathologic products in the fasting contents of two cases. No retention occurred in any case.

Group C.—Six cases. All were markedly depressed, some with depressing somatic delusions and four with considerable malnutrition. The gastric findings were not fully in accordance, though all cases showed either hypochlorhydria or moderate disturbance of motility.

REPORT OF CASES IN GROUP B

CASE 5.—*Manic-Depressive Psychosis, Depressed Type.*—Blanche D., a woman, aged 63, who was admitted Jan. 22, 1923, had had four previous periods of depression. The onset of the present attack occurred one month before admission. She had gas and belching one-half hour after meals, and experienced a "worried feeling" in the abdomen. There were no other dyspeptic symptoms, and the bowels were regular. Nutrition was good, and she weighed 163 pounds (74 kg.); the skin was yellowish. Blood pressure was 148 systolic, 86 diastolic; the urine showed occasionally hyaline casts, but no albumin. Basal

*Gastric Analyses in Forty-Six Cases of Mental Disorder**

Case	Fasting Contents					Fractional Examination of Gastric Contents												
	Quantity, C.c.	Acidity		C.c. N/10 NaOH		1 Hour Acidity		1 Hour Pepsin	Maximum Acidity			Curve Type	Pancreatic Ferments				Regurgitation Bile	Evacuation Time in Hours	
		Total	Free	Trypsin	Lipase	Total	Free		Total	Free	Time in Hours		Trypsin	Time	Lipase	Time			
A. 1	48	21	13	...	...	61	53	N	71	60	1¼	N	.....	.....	.....	.....	0	3	Pancreatic ferments present throughout digestion in another test
2	12	4	0	...	...	66	40	N	85	65	1¼	N	.....	.....	.....	.....	Fasting	2½	Pancreatic ferments present in another test
3	32	27	19	0	0	64	36	N	70	50	1¼	N	0	.....	0	.....	0	3	
4	14	19	11	0	0	61	40	N	80	41	1¾	N	10-23	End	10-15	End	End	2½	
B. 5	10	4	0	104	244	4	0	0	11	0	1¾	S	7-24	Throughout	7-49	Throughout	0	2½	Lactic acid positive
6	12	2	0	50	70	4	0	0	5	0	2¼	S	21-41	Throughout	50-150	Throughout	0	2¼	Maximum acidity in another test, 26
7	50	4	0	30	180	8	0	Tr.	19	0	2	S	30-130	Throughout	10-420	Throughout	0	2¼	
8	10	7	0	35	0	12	0	Tr.	16	0	2¼	S	15	End	0	.....	0	3	
9	6	15	5	175	540	10	0	Tr.	27	18	2¼	D	15	Beginning	30	Beginning	Beginning and end	2½	
10	15	7	0	0	20	25	7	Tr.	30	17	1¾	D	15	End	20	End	Fasting	2¼	
C. 11	15	6	0	0	0	8	0	Tr.	13	0	2	S	10-50	Throughout	10	Middle	0	2¼	Acidity in another test, 28-0; maximum, 37-7; delayed curve
12	25	6	0	110	305	28	16	Tr.	28	16	1	S	10-110	Throughout	13-535	Throughout	Fasting and end	2	Acidity in another test, 35-16; maximum 54-36
13	45	18	0	129	202	49	11	N	58	17	1¾	D	10-20	End	20-75	Beginning	Fasting	3+	Acidity in another test, 22-0; residue at 2¾ hours, 92 c.c.
14	15	10	0	157	240	45	24	N	49	40	2	N	40-120	End	4-154	End	Beginning and end	2½	Acidity in another test, 11-0; maximum, 48-33; delayed curve
15	15	11	0	85	440	54	30	N	54	30	1	N-C	10	Beginning	40-180	Beginning	Fasting and beginning	3	Acidity in another test, 38-8; more depressed period; residue at 2¾ hours, 55 c.c.
16	..	..	..	....	...	63	24	N	63	24	1	N-C	.....	.....	.....	.....	End	3+	Residue at 2¾ hours, 195 c.c. fluid and food
D. 17	10	16	0	...	...	42	13	N	..	..	...	N	.....	.....	.....	.....	End	2½	
18	36	20	0	...	...	57	27	N	..	..	...	N	.....	.....	.....	.....	End	2¼	
19	14	4	0	...	...	54	30	N	54	30	1	N-C	.....	.....	.....	.....	Beginning	2¾	
20	20	13	0	30	90	56	32	N	56	32	1	N	20-95	Beginning and end	10-310	Beginning and end	Beginning and end	2½	
21	9	4	0	0	0	42	7	N	52	31	2	N	0	.....	0	.....	None	3+	Residue at 3 hours, 35 c.c. fluid and food

E. 22	10+	10	0	...	...	62	35	N	82	62	1¼	N	.....	.....	.....	.....	None	3	
23	8+	11	0	215	605	61	25	N	76	54	2½	D	45-65	Beginning	105-145	Beginning	Fasting	2¾	
24	48	7	0	15	370	21	0	0	32	0	2¼	S	10-90	End	30-250	Throughout	End	2¾	Acidity in another test, 66-33; maximum, 67-46; delayed evacuation
25	..	..	..	...	...	68	45	N	68	45	1 1/12	N	.....	.....	.....	.....	End	2½	Residue at 2 hours, 75 c.c. fluid and food
26	10+	64	38	40	20	82	50	N	..	..	...	C ?	.....	.....	.....	.....	.....	...	Fasting contents and 1 hour Ewald test only
27	..	..	..	...	...	76	53	..	81	55	1½	C ?	.....	.....	.....	.....	0	2¾	
28	37	91	81	0	0	88	76	N	109	99	1½	C	25-40	Beginning	20-40	Beginning	Fasting	3	
F. 29	3	4	0	280	230	7	0	Tr.	18	0	½	S	62-254	Throughout	42-232	Throughout	Beginning, end and fasting	1¾	
30	20	24	17	30	10	12	6	Tr.	40	34	1½	D	8-223	Throughout	10-1240	Throughout	Fasting and end	2½	
31	..	..	..	...	...	34	14	..	59	26	1¾	D	.....	.....	.....	.....	0	1¾	
32	..	..	..	...	...	56	27	..	56	27	1¼	N	.....	.....	.....	.....	End	2½	
G. 33	..	..	..	...	..	19	12	..	19	12	1	S	.....	.....	.....	.....	Throughout	2¾	
34	15+	19	10	25	85	33	21	N	42	34	2½	D	0	.....	0	.....	0	2½	Acidity in another test, 56-31 at 1 hour
35	6+	2	0	180	225	24	12	Tr.	30	16	2	D	30-80	Throughout	20-120	Throughout	0	3+	Residue at 2¾ hours, 70 c.c. food and fluid; acidity in another test, 46-17; maximum, 50-19; delayed evacuation
36	10+	22	9	225	35	37	28	Tr.	37	28	1	S	25-125	Throughout	40-180	Throughout	0	2¼	Acidity in another test, 51-28; maximum, 79-64
H. 37	28	34	26	45	20	40	19	Tr.	57	26	1½	D	15-55	End	10-40	End	End	2¾+	
38	38	69	53	...	...	66	54	N	85	74	½	C	.....	.....	.....	.....	0	2+	
39	10	21	16	0	0	65	45	N	65	45	1	N	0	.....	20	End	0	2¼	
40	..	..	..	...	...	72	45	..	72	45	1¼	N	.....	.....	.....	.....	0	2½	
41	14	32	22	0	0	54	45	N	88	75	2½	C	0	.....	0	.....	End	2½+	
I. 42	..	..	..	...	...	30	13	Tr.	36	22	1½	L	.....	.....	.....	.....	Throughout	3	Residue at 2 hours, 152 c.c. food and fluid
43	22	58	42	0	0	73	31	N	90	78	1¼	C	0	.....	0	.....	0	3	
44	..	..	..	...	...	50	42	..	60	32	1¼	N	.....	.....	.....	.....	0	2¼	
45	..	..	..	...	...	60	47	N	65	48	1½	N	.....	.....	.....	.....	0	2½	
46	17	69	60	0	35	76	49	N	76	49	¾	C	50	End	5-35	Throughout	0	2½	Residuum following lavage showed 53 c.c. food and liquid at 2½ hours

* One or more confirmatory tests were made in each case; the results are not noted unless they were markedly divergent from the charted figures. N, normal; Tr., trace or diminished; S, subacid or anacid; D, delayed acid response; L, early acid response (larval); C, continuous secretion.

metabolic rating, 37.3 or 8 per cent. plus (limit of normal). Emotionally she was depressed, anxious, and retarded.

The gastric findings were: acidity at the fasting level, 4 to 5; lactic acid present; pepsin absent; duodenal ferments present but low; evacuation prompt. There is a gastric achylia, which may be organic (dyspeptic history), or possibly related to the psychic depression.

CASE 6. *Involutional Melancholia.*—Bertha A., a woman, aged 54, who was admitted Oct. 27, 1920, and had now been ill three years. She was self accusatory, apprehensive, irritable, confused, and hypochondriacal, with somatic and depressive delusions. She weighed 104 pounds (47 kg.) and was emaciated and pale. Her appetite was fair; the bowels were regular but with recurrent attacks of diarrhea (lientery). The abdominal walls were thin, with visible intestinal coils, but no definite mass. The basal metabolic rate was 29.7 (21 per cent. minus). Tuberculosis and carcinoma were suspected, but apparently discounted by the general improvement.

The gastric findings of achylia, prompt evacuation and history of recurrent diarrhea suggest organic changes in the mucosa with compensatory duodenal ferments in the stomach. Relation to the depressive psychosis is, however, possible.

CASE 7. *Manic-Depressive Psychosis, Depressed Type.*—Betsie W., a woman, aged 49, who was admitted Oct. 17, 1922, had had a previous attack in 1909. The present attack began six weeks before admission. The patient was agitated, apprehensive and suicidal. At the first test she was moderately resistive; at the second she spoke repeatedly of being lost. She had had constipation, anorexia, moderate loss of weight, from 176 to 123 pounds (80 to 55 kg.). The eyelids were full; the pupils were unequal and the right did not react; the skin was moist; the lower molars were lost, and pyorrhea was present. There was a nasopharyngeal catarrh. In this patient there was marked mental depression combined with loss of nutrition.

The gastric findings were: achlorhydria, low pepsin with low fasting level (4), and early evacuation of the stomach. The duodenal ferments were present in considerable amounts, both in the fasting stomach and after the test meal.

CASE 8. *Manic-Depressive Psychosis, Depressed Type with Catatonia.*—Belinda B., a woman, aged 56, who was admitted Dec. 21, 1922, had had fourteen previous attacks, the last in 1919. The present attack began in Octber, 1920. She had been agitated and apprehensive and imagined that she was reduced to an "atom." She was thin and anemic; had thin gray hair, and her teeth were poor. She lay curled up in bed with knees against her chin. She was uncommunicative, but keenly appreciative of her surroundings. She resisted throughout the tests. Urine examination revealed: trace of albumin, leukocytes and occasional hyaline casts; the blood count was only slightly reduced (4,200,000).

The gastric findings were: achlorhydria, almost complete lack of pepsin, very low duodenal ferments, no lipase and delayed evacuation. Here again physical conditions, cachexia, etc., are possible factors.

CASE 9. *Involutional Melancholia.*—Bess S., a woman, aged 55, was admitted Nov. 12, 1921. The onset of the present illness was in September, 1921. At the time of examination she was depressed, sullen and resistive. She had had a "sluggish liver" for many years, and gave a history of diarrhea on excitement in early life. Since admission appetite had been variable. She had been frequently tube fed, and also had recurrent diarrhea. She had no teeth and was emaciated. The urine occasionally contained a trace of albumin, and indican

was increased; blood urea was 18.3 mg., nonprotein nitrogen 29.2 mg. per 100 c.c.

The gastric findings were: achlorhydria at one hour; later, hypochlorhydria with reduced pepsin, i. e., delayed curve; normal evacuation time; regurgitation of bile in the fasting stomach and at two and one-half hours; pus and epithelial cells in the fasting contents, probably from the nasopharynx; duodenal ferments normal in the fasting contents. The gastric findings correlated with emotional depression, subnutrition, the history of recurrent diarrhea, indicanuria, etc.

CASE 10. *Manic-Depressive Insanity, Depressed Type (Possible Dementia Praecox.*—Brunhilde H., a woman, aged 43, was admitted Feb. 11, 1919. The present attack began in December, 1918, during convalescence from an operation, and she had remained absolutely unresponsive and uncooperative. She resisted the tests. She had had slight stomach trouble and constipation all her life. Physical examination revealed: moderate emaciation, flabby musculature, impacted feces and dilated colon. Appendicostomy was performed, and was followed by medical treatment. The bowels became normal. She had to be spoon or tube fed. The urine contained a faint trace of albumin, a trace or more of indican, and occasional hyalin casts; there was slight reduction of red blood cells (4,000,000), and the hemoglobin was 59 per cent.

The gastric findings were: the fasting contents contained mucus, and epithelial and pus cells; low acid and low duodenal ferments; gastric secretory curve delayed; hypochlorhydria and diminished pepsin; early emptying. The gastric findings are correlated with anxious depression, depraved nutrition and the gastro-intestinal pathologic conditions; they suggest a possible gastritis.

REPORT OF CASES IN GROUP C

CASE 11.—*Involutional Melancholia.*—Celia K., a woman, aged 52, who was admitted Oct. 18, 1919. The onset of the present attack occurred at 48. At the time of the test she was depressed but cooperative and quiet. She required to be spoon fed, and was constipated and poorly nourished. Physical examination was negative. There were transient albuminuria, low urinary specific gravity, hyalin and granular casts; the blood count was practically normal; basal metabolic rate, 35 (normal).

The gastric findings were: hypochlorhydria or achlorhydria with delayed curve, diminished pepsin; low or no duodenal ferment in the fasting stomach and during digestion; early emptying. These findings are correlated with a depressed mental state and depraved nutrition. Secretion appears to have been inhibited while motility was normal or increased.

CASE 12.—*Manic-Depressive Psychosis, Involutional Type.*—Catharine H., a woman, aged 53, was admitted May 28, 1922, with a history of onset of the illness nine months before. A month later she began to say she had no internal organs, no stomach, no taste, no smell, "everything gone." She had to be tube fed. She lost 48 pounds and weighed only 86 pounds (40 kg.) on admission. The general physical examination was negative. Urine examination revealed: slight glycosuria, no albumin, an occasional granular cast.

In this patient gastric tests revealed: normal acidity with delayed curve and in a second test hypochlorhydria; normal gastric and duodenal ferments; early evacuation. The nasal route was used. The findings are correlated with mental depression, somatic gastro-intestinal delusions, and subnutrition.

CASE 13. *Involutional Melancholia.*—Clara D., a woman, aged 50, was admitted Jan. 15, 1923, with a history of onset of the illness three months before. She had an agitated depression with vague somatic delusions, e. g., that her throat was eaten out, that she was half alive and could never get well. She cooperated fairly well. She was tall and undernourished. Saliva was increased; urine at first showed marked albuminuria with hyalin casts.

The gastric findings were: in the first test achlorhydria due in part to retching and duodenal regurgitation; in subsequent test normal acidity with somewhat delayed curve; much mucus; pepsin normal; trypsin and lipase low except in fasting contents; motility slightly delayed. These findings are to be correlated with psychic depression and malnutrition.

CASE 14. *Manic-Depressive Psychosis, Depressed Type.*—Constance P., a woman, aged 35, was admitted Oct. 16, 1922, with a history of a similar attack twelve years before with the same somatic delusion of a rat in her stomach. Appendectomy had been performed at 24. The present attack began six weeks before admission. The patient had had a very few dyspeptic symptoms; she expectorated phlegm and sometimes had a gnawing sensation in the epigastrium shortly after eating. Her nutrition was good and there was no unusual dryness of the skin, etc. There was a moderate sized, asymmetrical goiter, but there were no general symptoms and no evidences of pressure, and no dysphagia. The pulse rate was 80; the blood pressure 110 systolic, 72 diastolic; basal metabolism on two occasions was normal (36.5).

The gastric findings were: in the first test, achlorhydria at one hour with delayed normal; later (coincident with clinical improvement) normal curve, acidity sustained longer than usual, ferments normal, evacuation normal. These findings are correlated with a depressing somatic delusion. Nutrition was good.

CASE 15.—*Manic-Depressive Psychosis, Depressed Type.*—Christina H., a woman, aged 60, was admitted Sept. 4, 1922, with a history of two previous attacks. The present attack began in November, 1921. At the first test she was resistive, at the second cooperative. She had anorexia, only ate because it was unavoidable. She felt that she was filled up, and had a sense of gas and distention in the stomach and bowels. The bowels were constipated; the skin, dry, wrinkled and sallow. She was emaciated, her weight being 71 pounds (32 kg.). Her upper teeth were gone and the lower in bad condition. Salivary secretion was apparently suppressed. The urine showed a trace of albumin and hyalin casts; a nephritic test meal showed that power of concentration was present; the blood count was normal; the Wassermann test negative; the blood pressure, 120 systolic, 84 diastolic (at first 200 systolic, 110 diastolic); basal metabolism, 35 (normal). The medical diagnosis was arteriosclerosis (arteriosclerotic kidney).

The gastric findings were: first test hypochlorhydria and delayed evacuation; subsequently, with clinical improvement, normal acidity became continous, normal ferments, delayed evacuation. Correlated with the severe emotional depression, obstipation, subnutrition, etc., are found delayed motility, and on one occasion achlorhydria.

CASE 16.—*Manic-Depressive Insanity, Depressed Type (Possibly Mixed).*—Cora C., a woman, aged 33, when admitted Nov. 28, 1922, was moderately well nourished but pallid. At the onset severe headaches had been experienced on account of which ten teeth were removed. She had repeatedly required tube feeding. She resented examination violently. There was an excess of ropy mucus at the first test. The urine showed a transient albuminuria.

The gastric findings were: one test, achlorhydria; another test, normal acidity at one hour, well sustained curve, normal ferments, delayed evacuation. These findings are to be correlated with severe mental depression and lack of cooperation.

SUMMARY OF CASES IN GROUPS D AND E

These cases belong to the same clinical groups (manic-depressive) as the preceding groups. The depression in four or five was very mild, in the remainder it was quite definite. Cooperation was good or fair in all but one. The latter resisted violently. Five of the patients were anxious or resentful; three had hypochondriacal ideas or delusions referable to the gastro-intestinal tract. Nutrition was good in eight, fair in two, very poor in two.

The five cases in Group *D* had normal acid values at one hour with normal pepsin in all; the seven cases in Group *E* showed acid values which we would formerly have called hyperchlorhydria. In one case there was continuous hypersecretion.

It is difficult to reconcile the clinical and laboratory findings in these cases; nevertheless, it is noteworthy that mental depression was less and nutrition better than in the previous groups.

REPORT OF CASES IN GROUP D

CASE 17. *Involutional Melancholia.*—Dora H., a woman, aged 47, was admitted Jan. 17, 1921, in a state of good nutrition. At the time of the tests she was mildly depressed and resentful. The tests showed normal fasting contents, normal acidity and curve, normal pepsin, and normal evacuation.

CASE 18. *Involutional Melancholia; Carcinoma of Descending Colon.*—Donna S., a woman, aged 45, was admitted March 1, 1923. She was anxious and depressed, but cooperative. Nutrition was good, but the diagnosis of cancer was subsequently confirmed by operation. Tests showed findings almost identical with those of Case 17.

CASE 19. *Involutional Melancholia, Convalescent.*—Della Y., a woman, aged 51, was admitted Aug. 23, 1922. She showed then little or no depression. There was low acidity in the fasting gastric contents; otherwise the findings were similar to previous cases.

CASE 20. *Manic-Depressive Psychosis, Involutional Type.*—David McC., a man, aged 60, was admitted Feb. 27, 1923, in a state of marked depression. He was cooperative. He expressed hypochondriac ideas about his stomach. He was extremely emaciated, and had been operated on for carcinoma previously with negative findings. He presented normal gastric findings corresponding to other members of the group.

CASE 21. *Manic-Depressive Psychosis, Depressed Type; Tendency to Catatonia.*—Dina G., a woman, aged 23, was admitted Oct. 17, 1922. She was depressed and saliva drooled from her month. There were: low acidity in the fasting stomach; normal acidity at one hour, rising later; moderate delay in evacuation.

REPORT OF CASES IN GROUP E

CASE 22. *Involutional Melancholia.*—Etta W., a woman, aged 68, was admitted Jan. 1, 1923. She was moderately depressed and anxious in the first test, but was more normal in the second. Acidity in the first test was low normal but delayed. In the second there was hyperchlorhydria, with a normal curve. Duodenal ferments were high in the fasting contents and after two hours; pepsin was normal, evacuation time normal.

CASE 23. *Involutional Melancholia.*—Ella C., a woman, aged 46, who was admitted Jan. 11, 1923, was well nourished, but in a fairly deep depression and not very cooperative. In the first test she gagged; there was much mucus, but normal acidity. In the second test acidity was normal at one hour; later there was hyperchlorhydria (86), i. e., there was a delayed curve. Pepsin was normal; duodenal ferments were normal in the fasting contents and at the end of digestion. Evacuation was normal.

CASE 24. *Manic-Depressive Psychosis, Depressed Type.*—Emma W., a woman, aged 31, who was admitted Dec. 10, 1922, was fat, sluggish, depressed and apprehensive, and had delusions of sinfulness. The first test revealed moderately high acidity, normal curve, and normal evacuation. The second test (taken when the patient was more depressed) showed hypochlorhydria, no pepsin at one hour; duodenal ferments were present at one-half hour; evacuation was normal or slightly delayed.

CASE 25. *Manic-Depressive Psychosis, Depressed Type.*—Edward F., a man, aged 61, was admitted Nov. 10, 1921. He was retarded and markedly depressed, but chewed tobacco. The tests were incomplete, but showed hyperchlorhydria, normal curve, early emptying.

CASE 26. *Manic-Depressive Psychosis, Depressed Type.*—Ethel R., a woman, aged 63, was admitted April 16, 1921. She resisted violently and had vague somatic delusions. She was given an Ewald test meal. There were found: hyperacidity in fasting contents, and hyperchlorhydria at one hour. There was probably a continuous secretion.

CASE 27. *Manic-Depressive Insanity, Depressed Type.*—Edith S., a woman, aged 54, was admitted Jan. 9, 1923. She was well nourished, with a mild depression, and had psoriasis. Gastric tests revealed hyperchlorhydria with a well sustained curve.

CASE 28. *Manic-Depressive Depression with Somatic Delusions Referable to the Gastro-Intestinal Tract.*—Edgar R., a man, aged 33, was admitted Feb. 1, 1923. He was depressed and suicidal. Gastric tests revealed: highly acid fasting contents; no duodenal ferments; marked hyperchlorhydria (maximum, 109). The form of secretion was continuous; at two and one-half hours there was much acid secretion remaining.

SUMMARY OF CASES IN GROUP F (PSYCHONEUROSES)

The four patients with psychoneuroses were all more or less depressed. One case showed achylia, probably organic; two hypochlorhydria; one slight hyperchlorhydria. No definite conclusion was drawn as to the effect of the psychic factor, but the findings are at least suggestive.

REPORT OF CASES IN GROUP F

CASE 29. *Psychoneurosis (Psychasthenia).*—Frank K., a man, aged 54, was admitted July 19, 1919. He was well nourished but apprehensive, and gave a history of gastro-intestinal disorder with recurrent diarrhea. Gastric studies revealed: complete achlorhydria in two tests, low pepsin, high duodenal ferments in the fasting sample and throughout the digestive phase, and early evacuation.

CASE 30. *Psychoneurosis (Hysteria).*—Freda S., a woman, aged 23, was admitted Feb. 24, 1923, in a state of fair nutrition; she was moderately depressed and emotional. Gastric examinations revealed: normal acidity and ferments in the fasting contents; delayed hypochlorhydria after the test meal; trace of pepsin; and high duodenal ferments almost throughout.

CASE 31. *Psychoneurosis (Hysteria).*—Fannie G., a girl, aged 16, was admitted Jan. 20, 1923. Gastric test revealed: hypochlorhydria, normal curve, and normal evacuation.

CASE 32. *Psychoneurosis (Psychasthenia).*—Faith P., a woman aged 36, was admitted Jan. 19, 1923. She made all sorts of neurotic complaints, and appeared to be depressed. Gastric examination revealed a larval hyperchlorhydria with normal evacuation. Only one test was made.

SUMMARY OF CASES IN GROUPS G AND H

Cases 33 to 36 (Group *G*) were examples of dementia praecox of the paranoid type. All presented emotional irritability or instability. In one case examination was unsatisfactory (the patient gagged and was flighty); there was hypochlorhydria, the evacuation time being normal. The remaining cases showed normal acidity at one hour with a tendency to higher figures later (delay in the curves); pepsin and duodenal ferments were normal. In two cases evacuation was delayed.

Cases 37 to 41 (Group *H*) were also examples of dementia praecox of other types and included one patient who was practically well. All five patients were quiet and cooperative. All showed decided hyperchlorhydria (high normal?) with normal pepsin; duodenal ferments were absent in two, present in one case; the evacuation time was normal. One case had retention and a continuous curve. The fasting contents showed a high acidity.

The case histories in these groups have been omitted; there was no discernible relation between the secretory curve and the emotional aspect.

SUMMARY OF CASES IN GROUP I

Cases 42 to 46 were examples of miscellaneous psychoses—general paralysis, toxic-exhaustive and senile psychoses. The general paralytic drooled and was irritable. The test was unsatisfactory but showed hypochlorhydria with delayed evacuation. The other patients were quiet, apathetic or slightly depressed. High fasting acidity was found in the two cases in which it was tested. Hyperchlorhydria was present

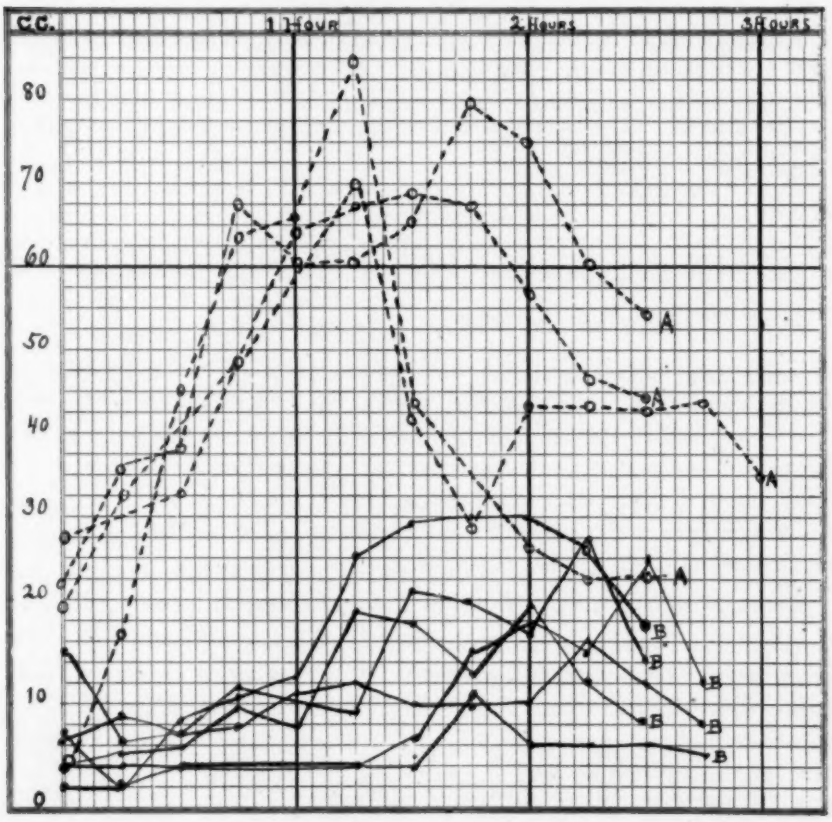
in all with continuous curves in two (in both with a tendency to retention). No definite relation to the emotional state was discernible.

SUMMARY

1. Manic-depressive psychoses and involutional melancholia.

(a) Manic States.—Four hypomanic cases showed a normal secretory response with a high acidity, unduly sustained. Formerly we would have described these cases without hesitation as hyperchlorhydria. The emotional state certainly did not interfere with secretion and possibly favored it (vagus action?).

(b) Depressive States.—Six cases showed marked reduction or delay in gastric secretion entirely consistent with the mental picture. Six similar cases showed less marked though still definite disturbance of gastric function. Five cases of similar clinical type, though on the whole less profoundly depressed, presented normal secretory findings.



Curves of total acidity. *A*, hypomanic types; *B*, depressed types. Cases 1 to 10.

Seven cases, including some with mild depression and others with presumably depressing somatic delusions, showed high acidities altogether comparable to those in the manic states.

Of the twenty-four depressed cases, approximately half showed distinct disturbances of secretion in the sense of diminution; in a few, motor disturbances occurred. So far we have been unable to distinguish satisfactorily between the effects of subacute or persistent psychic depression and of the acute temporary emotion associated with dislike for or resistance to the use of the tube. In several instances patients were examined by the nasal catheter and in a few mechanical restraint was used (pack). It is noteworthy, however, that the two patients who resisted most violently gave rather high acid values.

We feel that these observations show good evidence of the inhibiting effect (sympathetic) of profoundly depressing emotions and less definite evidence of the favorable effect (vagus) of elation. It is hardly necessary to add that psychiatrists regard the emotions in this group of insanities as real in contrast with the emotions in dementia praecox which are superficial, unreal and not in accord with the circumstances.

2. Psychoneuroses.—In these cases emotional depression was a uniform symptom, but the depth of the emotion was less pronounced and its sincerity less unquestionable than in the preceding depressive states. Only four cases were examined and, while reduced acidity was evident in three, in one at least there was a sufficient physical cause. Experience, moreover, warns against drawing too broad conclusions from this class of cases.

3. Dementia Praecox.—Of four paranoid cases with emotional irritability and instability two showed moderately delayed evacuation with normal acidity. Five other cases of dementia praecox all showed high acidity (hyperacidity), but no relation was detected between the emotional state and the gastric secretion.

4. Miscellaneous Psychoses.—Five cases of general paralytic, toxic-exhaustive or senile psychoses in which apathy or slight depression was the rule, showed high acidity in four and continuous secretion in two.

CONCLUSIONS

Depressing emotions appear to exert an inhibiting effect on gastric and even on duodenal secretions. Motility is less clearly influenced, but requires further investigation. Malnutrition may be a principal factor in causing reduced gastric secretion, but it is more likely that the malnutrition is secondary to the digestive disturbance.

Somatic and hypochondriacal delusions bear no evident relation to secretory variation. Mental exaltation seems to favor gastric digestion.

News and Comment

PARKINSON'S SHAKING PALSY

As only four copies of Parkinson's classic Essay on the Shaking Palsy (1817) are known to exist, at our request the American Medical Association Press has reproduced this work in facsimile. The edition is limited to 300 copies, each numbered, and the price is $1.25, the actual cost of production. This unique work is of interest to every neurologist and would make an appropriate gift for an internist.

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Abstracts from Current Literature

DISEASES OF THE STRIATE SYSTEM. Cécile and Oskar Vogt, J. f Psychol. u. Neurol. **25**:3, 1920.

This monograph, which takes up an entire number of the journal and consists of over 200 pages of text with 78 double pages of illustrations, is a sequel to a prior work by these authors on the same subject to which they refer extensively. The subject is taken up in a systematic manner. First, the normal anatomy is discussed and, in order to make clear what follows, it will be necessary to give a rather full description of Figures 1 and 2 which are taken from the article. As will be noted Fig. 2 is an enlargement of Fig. 1 at level "III."

At level I, besides the thalamic nuclei *l, vtl, vtm* and *aa*, in *mv*, in the most oral part, is seen what Friedmann has described in the cercopithecin as the nucleus of the tuber cinereum *t*. Ventral to *mv* lies the main part of the most oral portion of the field of Forel, H^2 with its mesial process *X* drawn out into the tuber cinereum. Then there are: the posterior part of the internal capsule *Cip;* the beginning of the peduncle *p;* and the mammillary body *Cm*.

At level II a section of the prefrontal cortex is represented.

At level III (which is enlarged in Fig. 2) is represented the thalamus in frontal plane, the corpus Luysi, the striatum and the pallidum. In the thalamus are seen the nuclei *l, aa, ma, vtl. vtm* and *mv*. In the hypothalamus are: the H^1 and H^2 bundles of Forel as well as their median junction; Cajal's nucleus campi Foreli (*ncf*); the pars dorsalis (*Zid*) and the pars ventralis (*Ziv*) of the zona incerta and the corpus Luysi (*CL*). In the peduncle (*P*) is the oral portion of the substantia nigra (*Sn*). At its medial part is found dorsally, the commissura mollis (*Cmo*) and ventrally the decussatio Foreli (*DF*). Dorsolateral to the thalamus is the nucleus caudatus, (*Nc*); lateral to the posterior segment of the internal capsule (*Cip*) is the putamen (*Put*) and the pallidum. The pallidum is bounded laterally by the lamella pallidi externa (*Le*); ventrally by the ansa lenticularis (*Al*) and medially by the lamella pallidi limitans (*Ll*). The lamella pallidi interna (*Li*) divides the pallidum into a pars externa (*Ge*) and a pars interna (*Gi*). The latter is divided by the lamella pallidi accessoria (*La*) into *Gil* and *Gim*.

At level IV there is represented a part of the area gigantopyramidalis, "the so-called motor region."

At level V is a frontal section of the hypothalamus with its nucleus ruber (*Nr*). Dorsally this region is bounded by the thalamic nuclei: *va, vb,* and *mb*. Medially is the decussation of Forel (*DF*). Ventrally is represented substantia nigra (*Sn*), *P* and the beginning of the pons. Beside this there is quite schematically represented a piece of the cerebellar cortex with its nucleus dentatus (*Dt*) of the opposite side.

Level VI represents the commissura posterior (*Cp*), the nucleus Darkschewitschi (*ND*) commissura posterioris (*Cp*) and Cajal's nucleus interstitialis (*ni*).

At level VII is represented the nucleus Bechterewi (*nB*) and the nucleus Deitersi (*nD*) as well as the canalis semicircularis horizontalis (*Csh*) and the canalis semicircularis verticalis anterior (*Csva*).

The structures represented are connected as follows: From the prefrontal cortex Fiber 1 goes to the thalamic nuclei *ma*; from *ma* is drawn an hypothetical

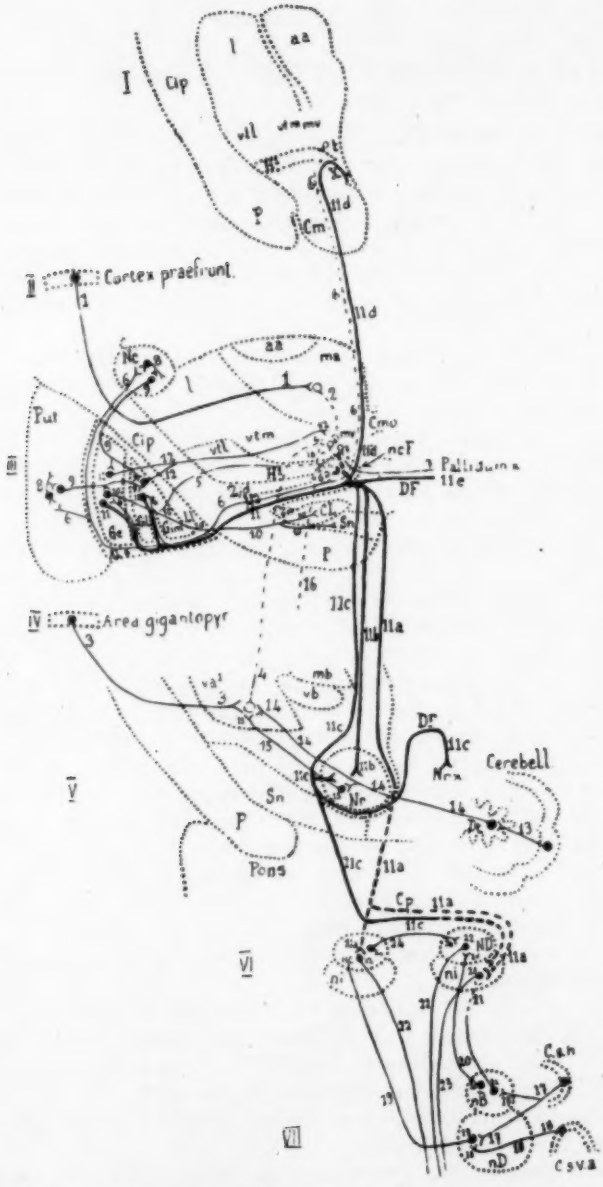

Fig. 1.—Diagram of connections of striatal and thalamic nuclei. The lettering is explained in the text.

associate Fiber 2 into the region of *mv* + *t* + *ncF*. From the motor cortex there goes a descending Fiber 3 to the thalamic nucleus *va*. From this is postulated a hypothetical intrathalamic Fiber 4 to unite with *mv* + *t* + *ncF*. From these latter regions Fibers 5 go as thalamopallidal and thalamostriatal through the thalamus as well as the hypothalamopallidal and thalamostriatal Fibers 6 through H^2 to the pallidum and striatum. Fiber 5 is ended in *Gim* exclusively so as not to complicate the picture. Besides, there also goes from the region of *mv* + *t* + *ncF* a Fiber 7 through *DF* to the opposite pallidum. While Fiber 6 ends in *Put* and *Nc*, it comes into relation with an association Fiber 8. This again stands in relation to Fiber 9 which sends striopallidal fibers to *Ge* and *Gi*. In the pallidum there arises a branch, Fiber 10, to *CL* (corpus Luysi) and in less degree to the substantia nigra (*Sn*). (In Fig. 2 this is represented by 10a.) A second branch, Fiber 11, runs from the pallidum through the ansa

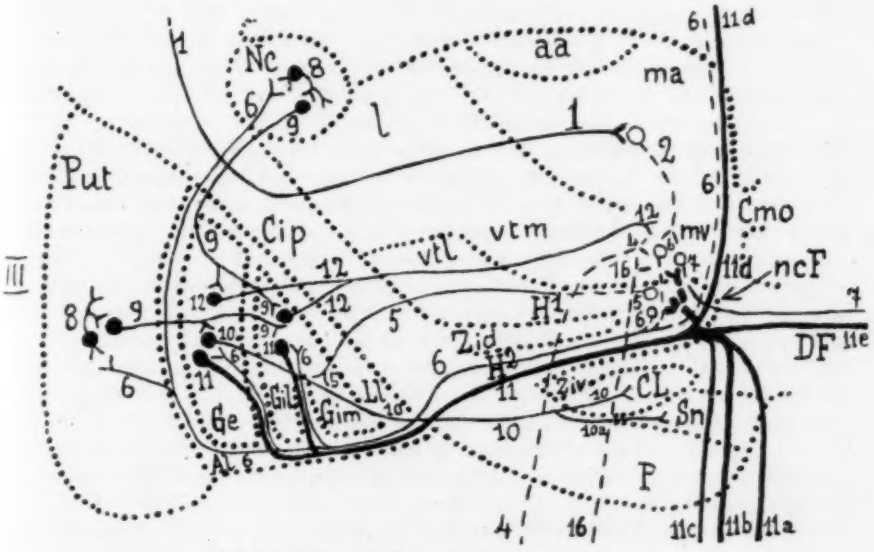

Fig. 2.—Enlargement of Level III in Figure 1.

lenticularis (*Al*) following H^2 and divides into five branches 11*a* to 11*e*. Branch 11*a* runs through the middle part of the capsule of the red nucleus; one part apparently ends in the nucleus Darkschewitschi (*ND*), and part goes through the posterior commissure to end in the opposite nucleus interstitialis *ni*. Branch 11*b* ends in the frontal part of the nucleus ruber; 11*c* helps first to build the lateral capsule of the red nucleus, and the fibers then go in part to this nucleus, and in part, after passing through the decussatio Foreli, to the opposite red nucleus, and in part to the posterior commissure. The last hypothetical portion is allowed to end in the opposite nucleus interstitialis, (*ni*). Branch 11*d* ends in the region of *mv* + *t* + *ncF*. Branch 11*e* goes, in the decussatio Foreli, to end on the opposite side. A third group of neurons sends its axons through the thalamus in the region of *mv*.

From the cortex cerebelli the axis cylinders of the Purkinje cells, Fiber 13, run to the dentatum (*Dt*). The Fibers 14 of the dentatum go in part to the

red nucleus and in part to *va*. The former find their way out also from the red nucleus as Fiber 15. In Fiber 16 there are as yet unknown fibers from the region of *mv* + *t* + *ncF*.

The horizontal semicircular canal (*Csh*) sends Fibers 17 to the nucleus Bechterewi (*nB*) and to nucleus Deitersi (*nD*). The anterior vertical semicircular canal (*Csva*) sends a system of Fibers 18 to (*nD*). From *nD* Fibers 19 go to the opposite *ND*. From *nB* they go to the homolateral *ND* by Fibers 20 and probably by Fiber 21 to the homolateral *ni*. From *ND* and *ni* a system of Fibers 22 (fasciculus commissuromedialis) and 23 (fasciculus interstitiospinalis) go downward in the tractus longitudinalis dorsalis. Finally we have, in the posterior commissure, commissural Fibers 24 between the two *ND*.

In the comments on the foregoing, two facts are brought out forcibly, which are to be found in all modern textbooks on neuro-anatomy: First, there is no fiber connection between the cortex and the opposite striatum; secondly, there is practically no difference in structure and origin between the caudatus and the putamen. In brief, therefore, the Vogts state that the striatum is a terminal organ. Fibers do not connect the cortex with it except indirectly as follows: the cortical fibers proceed to the thalamus, from the thalamus to the pallidum and then from the pallidum to the striatum. It is connected also with the periphery indirectly as follows: fibers from the periphery go to the thalamus; from the thalamus they go to the pallidum (and also to the cortex); those that end in the pallidum go to the striatum. Fibers beginning in the striatum go to the pallidum; from there they go through the ansa lenticularis and divide into branches to the thalamus and to the subthalamic nuclei, including the red nucleus, substantia nigra, corpus Luysi etc. From there the fibers proceed down into the cord as the rubrospinal tract, and in this way have a direct influence on the anterior horn cells. Finally the cerebellum is brought into relation with this system through fibers going to the red nucleus.

In the further discussion the authors state that the fiber system between the thalamus, hypothalamus and the pallidum is medullated for the greater part at the time of birth. On the other hand, the striopallidal fibers (Fiber 9 in Fig. 1) are not medullated even at 5 months. It is practically universally believed that beginning of medullation shows the beginning of function. In the new-born the striatum and pallidum do not communicate. The similarity between the movements of infants and those that occur in disease of the striatum—muscle spasm, associated movements, chorea, athetosis, etc.—becomes clear through a study of these facts.

The rest of the article is then divided into eight chapters and the following types of case are discussed: (1) état marbré (marbled condition); (2) stationary état fibreux (fibrous condition) as part of the Bielschowsky type of cerebral hemiatrophy; (3) progressive état fibreux; (4) état dysmyélinique; (5) total necrosis of the striatum; (6) areas of neuroglia proliferation in the striatum with simultaneous presenile changes in the striate system; (7) état de désintégration; and (8) large focal lesions.

ÉTAT MARBRÉ

Eight cases of état marbré (marbled condition) are discussed at length. The symptoms are typified by the first case, which has been used by C. Vogt in a previous article and is known as "Barre's case of Jacquel." In this case there was a typical double athetosis of the face and body without paralysis of sphincters or severe intelligence disturbances, with increased knee jerks and ankle clonus. A brother had had involuntary movements in the hands.

The patient was born asphyxiated. Speech was slower to develop than walking. The condition had undoubtedly been present since birth. In the last year of life there developed a slight right sided paralysis. Death occurred at 59, probably by suicide.

The pathology of this case was summarized as follows: A condition of état marbré with decreased volume of the striatum. This was especially pronounced in the caudal part of the putamen, with decrease in the number of striopallidal fibers in the pallidum externum; marked internal hydrocephalus; the number of "plaques fibromyéliniques" of the cortex was within normal limits; the area gigantopyramidalis, H_1^2 and the corpus Luysi as well as the cerebellar system were intact; the left pyramid showed a decrease in its fibers because of the capsular softening in the last year of life. Bielschowsky found amyloid particles in the dendrites of the cells of the pallidum.

On the basis of the location of the lesion in this and other cases C. Vogt has formulated the following localization of functions: Lesions producing disturbances of the bulbar muscles are located in the most oral part of the striatum; caudad are the centers that control the lower extremities; between these, the abdomen, chest and upper extremities are represented.

A case of tremor of one forearm is mentioned in which there was involvement of the middle part of the opposite putamen.

Symptomatology.—The first six cases showed the picture of état marbré in such isolated form that the authors believe they are able to map out a symptomatology that can be applied to other cases. In the foreground stands hyperkinesis. This manifests itself in spastic phenomena and it remains to be seen whether contractures ever result when the case is handled correctly. In many cases the spasm is only temporary. A second form of hyperkinesis shows choreic movements and movements resembling athetosis. These involuntary movements occur in the slighter cases of état marbré in contrast to the spastic phenomena. A true Babinski sign is not present. Uncontrollable laughing and crying are frequently found. Peripheral stimuli, intentional or emotional, increase the hyperkinesis. There is slowness and clumsiness of movement as well as lack of emotional expression of the face. There are also severe disturbances in articulation, phonation, mastication and deglutition.

The question as to whether these symptoms are dependent on hyperkinesis or akinesis is discussed at length. In all cases the disease was bilateral and began after birth with a tendency to improve up to the age of 5. The authors' cases show more marked involvement of the lower than the upper extremities as is usual also in Little's disease. Pathologically, they believe that their hypothetical leg center in the striatum was not more severely diseased than the arm area. They therefore believe that there is a greater brain compensation for arm movements due to a greater representation of the arm in the brain cortex. These cases usually show at first equal involvement of the arm and leg with greater improvement of the upper extremity. In the seven cases studied anatomically they believe that they have made an advance in the correct topography of the striatum. They give credit to Marie, Brissaud and others.

Pathophysiology.—The authors believe that no exact opinion can be given as to the functions of the various tracts since normal anatomy and physiology have not been definitely determined. They took up this subject in their former work. They believe from a study of one of their cases that the entire picture may be present clinically, but absent pathologically. All the symptoms must be brought into relation with the striate system. In their case there was no

reduction of fibers between the thalamus and *ncF* with the pallidum. The corpus Luysi showed but a slight volume reduction. The pallidum was greatly reduced in size, but this could be attributed to the loss of fibers of the striopallidal system. The authors believe that there was defective embryonic development and the disease was due to a functional disturbance of the striatum that was present even in the embryonic brain.

The slight and severe cases differ only quantitatively, the pathologic changes being parallel to the symptomatology, and the striatum has a definite topography. The conclusion reached is that the symptomatology of état marbré is to be attributed to the absence of striate function and that the hyperkinesis is an inhibitory symptom.

As the result of past and present work the Vogts believe that they are able to formulate the following: Through Fibers 16, 14, 15 and others not represented (Figs. 1 and 2) impulses are carried to the thalamus. Part of these stimuli are direct (16) and others are indirect (through 4 and other associated neurons) going by way of Fibers 5 and 6 to the pallidum. Normally the striatum receives regular impulses which influence the pallidum through Fibers 8 and 9. If these impulses fail, impulses come from the periphery through the thalamus to the pallidum and especially by way of the corpus Luysi without the restraining effect of the striatum, and hyperkinesis results. This view explains why peripheral stimuli increase kinesis. The entire cortex is connected with the thalamus through the corticifugal Fibers 1 and 3, and in this way impulses can affect the pallidum through the thalamus. Cortical influences either "intentional" or "emotional" can increase pallidal kinesis. "In certain isolated motility disturbances it is difficult to know whether they are hyperkinetic, akinetic or a mixture of these two; the history does not help. How far the motor weakness can be traced to spasticity is problematic, but most cases of état marbré have a positive motor asthenia. Babinski has described, under the term adiadokokinesis, a slowness and uncertainty of antagonistic movements; for example, in pronation and supination of the hands. The patient may do it a little faster with the right hand and there may be a lessening of excursion of the left hand. This depends on an insufficiently prompt stimulus or "denervation," and occurs in greater degree in cerebellar disease. The Vogts here postulate a true adiadokokinesis depending on disturbance of voluntary denervation, in relation with the pallidum. They call pseudo-adiadokokinesis an "innervation" symptom consisting of slowness and clumsiness of movement; it plays an important part in état marbré and is atributed to absence of "kinesis" of the striate system. These movements in this condition may be due in part to the spasticity. The fact that hyperkinesis and akinesis of état marbré improve with age is explained by the statement that there is an increase of "innervation performing" ability of the brain which in time compensates.

Pathologic Anatomy and Genesis.—As shown in the Vogt's former work état marbré is an entity as far as the pathologic anatomy in concerned. The ganglion cells disappear and are replaced by a dense fiber network. Only in C. Vogt's cortical "plaques fibromyéliniques" do they find an analogous condition. They state that there are practically no "normal" human brains in which these plaques do not occur; yet they are not numerous. They have even found them, though less frequently, in monkey brains. If the plaques fibromyéliniques of the cortex and état marbré of the striatum can be attributed to the same cause the conclusion must be formulated that the striatum is much more liable to this condition.

The fact that this disease more often attacks brothers and sisters without the parents being affected points to an early germ plasm defect, and it is possible that the same condition may develop even after embryonic life. In one case described there was a true fibromyélinique condition with partial microgyria and meningitis, all of which are attributed to embryonic maldevelopment. The condition of état marbré shows a predilection for the inner half of the striatum.

ÉTAT FIBREUX

Elective necrosis of ganglion cells and the finest nerve fibers may occur in the striatum with crowding together of the remaining large medullated fibers. This results in a striking picture which C. Vogt has called "état fibreux." Four different etiologic forms of état fibreux are demonstrable in recognizable striate motility disturbances: (1) As part of the Bielschowsky type of cerebral hemiatrophy (e. g. spastic diplegia); (2) isolated; (3) as part of the process in Huntington's chorea; and (4) as a sequence of general paralytic processes in the striatum. The first of these is regarded as a stationary infantile condition; the other three appear later in life and are progressive.

STATIONARY INFANTILE ÉTAT FIBREUX

This is illustrated by the case of Fritz G., an illegitimate child, who, at the age of 2 began to have epileptic attacks, and in the course of the third year developed weakness of the left side and pes equinus, with marked athetotic disturbances of the hand, but without marked spasticity. There was no progression in the paresis up to the time of death at 21 from pneumonia.

Anatomic Findings.—In the brain, there were primary encephalitic foci leading to a hemiatrophy, the basis of which, as shown by Bielschowsky, was partial necrosis of the third cortical layer. There was also marked atrophy of the thalamus, but the brain stem was normal and there was no recognizable change in the pyramidal tracts. The elective necrosis in this case was not limited to the third cortical layer. The cell necrosis of the outer part of the caudate nucleus was so severe that the fibers remaining formed an état fibreux. In the rest of the striatum there was also an état fibreux. On the other hand the cell picture of the pallidum, the corpus Luysi, and the H^2 bundle showed no change. It must, however, be added that the neuroglia reaction in this case was not identical with that to be described as état fibreux.

In view of these findings the athetotic movements are traced to the état fibreux of the caudatus; the right thalamus had no part in the functional disability. In this case, the relatively slight manifestations of one sided paralysis are brought into relation with disease of the third cortical layer and the authors believe that the mechanism is similar to that shown by Bielschowsky. Whether the spastic paralysis indicated a striatal component or can be explained entirely by the cortical injury must be left to further work. In this case, as shown by Bielschowsky, there was an elective cell necrosis of the striatum. Sections from the cases of Bielschowsky showed changes resembling those described here but with more marked atrophy of one hemisphere and thalamus.

In relation to these facts the Vogts discuss:

(a) The rôle of the corticospinal system in the striate syndrome. In the two cases described by Bielschowsky there was spastic hemiplegia with no apparent striate symptoms. This is interpreted as indicating that the severe

cortical disease masked the striatal symptoms. In Fritz G. the slight cortical disease caused the striate symptoms to stand out. The conclusion reached is that the masking of the striate syndrome stands in direct relation to the intensity of the corticomotor disturbances.

(b) Von Economo stated that, in children as well as in the aged, after injuries of the brain in different locations athetosis may result. Economo gives credit for this observation to Magendie, in 1823; none of the cases, however, were studied with modern methods. The case of Fritz G. gives a clue as to how athetosis and a brain disease can occur together. The hemiatrophy was noticeable to the naked eye, but the striate lesions required study in serial sections with modern methods. The conclusion reached is that syndromes said to be due to cortical changes may, on further study, prove to be striatal.

(c) The production by striate disease at different times of athetosis, chorea, paralysis agitans, and other hyperkineses. This observation has puzzled many authors. Kleist recently offered the explanation that in athetosis the pallidum is diseased. The Vogts show that état fibreux causes, at the least, a severe secondary change in the pallidum and yet, in spite of this, may produce chorea. No cases are known in which disease limited to the striatum in fetal or in the first years of life has produced a chorea. The conclusion is reached that striate disease must occur in the embryo or in the first years of life to produce a true athetosis. Occurring later, if limited to the striatum, it occasions other involuntary movements.

(d) Etat fibreux as a sign of especial vulnerability of the striate cells. At present the three known cases of the Bielschowsky type of cerebral hemiatrophy show, in addition to elective necrosis of the third layer of cortical cells, a disease of the striate cells. Bielschowsky has investigated this question and comes to the conclusion that the elective necrosis of the third layer is due to a special vulnerability of these cells. Later the Vogts give further reasons for suggesting that the striatal cells are also especially sensitive.

(e) Part of the caudate is related to the arm muscles and the most anterior part has connections with the bulbar muscles.

(f) The clinical picture of état fibreux of the striatum as part of the Bielschowsky type of cerebral infantile spastic paralysis. At present the striate component cannot be certainly separated from the spastic manifestations. The slightly spastic cases are characterized in the beginning by epileptic spells, after several months by spastic hemiplegia, and later in life by hypoplasia of the musculature and skeleton. In the three cases quoted the disease was unilateral. Bilateral involvement or diplegia is possible. That other forms of cerebral spastic paralysis may be associated with disease of the striatum must be kept in mind, though the authors have not observed a case.

PROGRESSIVE ÉTAT FIBREUX

1. *Etat Fibreux Alone.*—Three cases are presented. The first concerns a progressive bilateral chorea of the body musculature in a man of 31 who for three and one-half years suffered with difficulty in articulation and walking. There was no hereditary taint, nor disturbance of eye movements, motor power or psychic power, but the knee jerks were increased. Anatomically this case showed pronounced chronic disease (atrophy) in the striatum, with much less change in the pallidum and still less in the corpus Luysi. There was a marked loss of ganglion cells, especially of the smaller type, with secondary glial increase and, according to Bielschowsky, a secondary reaction of the blood vessels (thickening of the vessel walls, capillary fibrosis and new capillary forma-

tion). The preservation of the medullated fibers in the striatum (Fiber 6 in Fig. 1) produced an état fibreux. The atrophy of the pallidum depended on loss of the fine fibers (Fiber 9 in Fig. 1) connecting the striatum with the pallidum. There was also primary necrosis of the striatal neurons (Fibers 6 and 9 in Fig. 1).

In commenting on this case the authors attribute the chorea to disease of the striatum. The severe atrophy of the oral region of the striatum explains the severe bulbar symptoms. The fact that the pallidum is more severely diseased than in their cases of état marbré brings up Kleist's remark that coincident disease of the pallidum in a case of état marbré produces athetosis in place of chorea. But against this is the conclusion that choreic movements are to be thought of as "the syndrome of the corpus striatum."

Bielschowsky's Case E.: A woman, aged 65, showed severe motor unrest involving the entire body; she rolled about in bed and moved the extremities in all directions. There was no intelligence defect. Pathologically, there was extreme reduction in the size of the striatum with an état fibreux and slight atrophy of the pallidum as well as a slighter decrease in the corpus Luysi. In commenting on this case the authors say "here, as in the previous case, the striatum is the chief seat of disease and we trace the chorea to this pathologic finding. Our views on the physiopathology of chorea as well as the objections to the theories of Kleist and von Economo, expressed in the preceding case we hold to be correct for this case."

Schuster's Case Heinrich N.: A case of chronic chorea with no history of hereditary defect, beginning at the age of 53 and lasting ten years. There was involvement of the eye musculature. Some intelligence defect was present. The brain showed marked état fibreux in the severely atrophied striatum, with much less atrophy of the pallidum and still less of the corpus Luysi.

Comments: In the adult central nervous system there occurs an isolated necrosis of the ganglion cells (especially the smaller type) of the striatum, with resultant reaction of the glia and blood vessels, and proliferation of the medullated fibers that remain. This causes an état fibreux with much less atrophy of the pallidum and still less marked atrophy of the corpus Luysi from secondary degeneration. The process is always bilateral. Clinically it is a progressive bilateral chorea. The authors have reported three cases and after observing the first case they were able to predict the pathologic changes in the others. The etiology is unknown. They have not found similar conditions except in chorea.

2. *Etat Fibreux Together with Typical Cerebral Disease.*—Freund's case, H. B., was typical of hereditary Huntington's chorea with mental deterioration. The anatomic findings were: widespread gliosis after disappearance of the nervous parenchyma especially of the ganglion cells. The process was largely limited to the striatum which was atrophied and presented the picture of an état fibreux. The pallidum was moderately atrophied from secondary degeneration and the corpus Luysi was small. The cortex cerebri was also involved in the disease process, especially the fourth layer. The Vogts "see the cause of the chorea in the disease of the striatal system." They refer to the work of Marie and Lhermitte in 1914, and of Hunt with four cases of Huntington's chorea in 1916, in which the only lesion was disappearance of the small striatal cells, but maintain "that the limitation of the disease process to the small ganglion cells is not correct." They conclude that the cortical changes account for the psychotic disturbances.

Liepmann's Case, Julie R.: Mental symptoms began at the age of 34 and soon there developed the typical picture of Huntington's chorea. No hereditary taint was present. Pathologically, in addition to congenital micromyelia, there was widespread parenchymatous degeneration with consequent gliosis. This was most intense in the striatum. In the pallidum there was a moderate secondary degeneration of the outgoing bundles and the thicker medullated fibers of the pallidum externum were of abnormal caliber. The corpus Luysi was likewise atrophied. H^2 and H^1 were slightly reduced. The cortex was also involved, especially the fourth layer. In the discussion, the chorea is brought into relation with the striatal disease. The extreme involvement of the oral part of the striatum is in line with the thought that this region controls the bulbar functions. The cortical changes produced the mental symptoms.

Maass' Case, Poersch: A woman, aged 71, whose history could not be obtained; she developed choreiform movements and dementia in the last years of life. Anatomically, the cortex showed narrowing and crowding together of the cells, decrease of the ganglion cells and, in the fourth layer, a great increase of glia. The striatum was distinctly small and showed an undoubted état fibreux. This atrophy had brought about an internal hydrocephalus. The pallidum was much atrophied and the corpus Luysi also to a much less degree. The internal capsule showed no fiber decrease. In addition the brain, especially the striatum and pallidum, showed an état criblé. In discussing this case the authors state: "There were the characteristic findings of Huntington's chorea in the striatum and the cortex. The état criblé did not reveal itself because of the état fibreux." Two other cases of Huntington's chorea are detailed.

Comments on Cases of Huntington's Chorea: These cases all show severe atrophy of the striatum, due to disappearance of the ganglion cells and compensatory increase of glia. The preservation and pressing together of the large medullated fibers of the striatum produces an état fibreux. There results internal hydrocephalus with granular walls. Less intense atrophy with gliosis occurs in the pallidum, and there is a decrease of the corpus Luysi. The brain is small with lessened density of layers and there is gliosis in the region of the fourth layer. There is decrease in the rest of the central nervous system with a similar gliosis. Clinically, all cases showed chorea together with psychotic disturbances. The pathologic findings in the striatum and cortex are diagnostic.

3. *Etat Fibreux as a Result of General Paralytic Disease Involving the Striatum.*—In Liepmann's case, Karl R., choreic movements were present during the last ten years of life. The facial muscles were involved as well as the extremities, though speech was but little affected. Two years after the beginning of the chorea there was sluggishness of the pupils and other symptoms of general paralysis of the insane. Anatomically, the cyto-architecture of the cortex was relatively normal. The chief abnormality was infiltration of the vessel walls. Bielschowsky showed that the vascular changes and cell characters were typical of general paralysis. The striatum showed considerable atrophy due to disappearance of the ganglion cells, while the thick medullated fibers were relatively well preserved. There was here a general paralytic process with elective necrosis of the ganglion cells which produced an état fibreux. There was increase of neuroglia nuclei, especially in the putamen, but not so marked as in the cases of Huntington's chorea. In addition there were general paralytic changes in the vessels. The pallidum showed, besides atrophy and general paralytic changes in the vessels, a cell decrease and disease of the

remaining ganglion cells that were more intense than was found in the cases of état fibreux up to this time. The usual thick medullated fibers of the pallidum externum showed pathologic thickening. The corpus Luysi was slightly small. The other subcortical regions showed no changes.

Comments: There is some doubt as to whether the intellectual defect was due to general paralysis. The anatomic studies showed the general paralytic process, but it is important to recognize that in this case, in spite of the long drawn out illness, the cortex was relatively intact, which explains the absence of the usual psychic disturbances of general paralysis. The cause of the chorea is found in the necrobiosis of the ganglion cells of the striatum. The disease of the pallidum needs explanation. The fact that the pallidum is diseased shows that this is possible without changing the choreic character of the involuntary movements. Further, it is interesting that the choreic movements entirely disappeared as the disease ran its course. Two important things are brought up by this case: (1) In rare cases of general paralysis the process attacks the striatum first and foremost; (2) in some cases of general paralysis the striatum and not the cortex is attacked. There is seen in these facts further proof of the especial vulnerability of the ganglion cells of the striatum.

Reich's Case, George M.: The patient was a man, aged 37, who had typical general paralysis in the last years of life. Two and one-half weeks before death, a violent chorea began. Pathologically, besides the marked general paralytic findings in the cortex, there were numerous typical vascular changes in the putamen. The same condition was found in the pallidum, but of much less degree. There was also a partial focal elective necrosis of the cells of the striatum. This produced first slight atrophy and then an état fibreux in the striatum. In contrast to the severe changes in the striatum, they were much less severe in the pallidum and corpus Luysi and only slight deviations from the normal were found in the other subcortical regions. The beginning elective cell necrosis in the striatum agrees with the clinical fact of chorea a few weeks prior to death.

4. *General Comments on the Cases Cited.*—All these cases have in common progressive cell necrosis in the striatum and general compensatory proliferation of neuroglia, which left no spaces and did not result in severe striatal atrophy. In this disease the majority of the thicker (striopetal) fibers remained, while the other fibers of the striatum were abnormally medullated. There thus resulted an état fibreux, the ganglion cell necrosis occurring when the condition had lasted for some little time. Practically identical pathologic changes resulted from at least three dissimilar causes. Each of the three forms of progressive état fibreux shows a specific staining: Huntington's chorea shows marked gliosis; and the general paralytic cases show the typical vascular changes. Of importance is the fact that almost identical changes occur in the striatum in progressive bilateral chorea, in Huntington's chorea and in general paralysis of the insane. The disease was so severe in the striatum and so slight in the pallidum and corpus Luysi that the authors trace the choreic movements to inhibition of the pallidum.

ÉTAT DYSMYÉLINIQUE

Under the term état dysmyélinique, C. Vogt has grouped cases in which pathologically there results simultaneous reduction in volume and poverty of striatal medullated fibers; this occurs especially in the region of the pallidum.

The two cases described give the picture of progressive, pure rigidity. The connection between the pathologic picture and the clinical disease is shown in Oscar Fischer's case described in their former work. The correlation between the cases that occurred in earliest infancy and those in juvenility is shown in the following case quoted from Rothmann. The patient was a girl who appeared to be normal at birth, but was slow in walking and talking and died at the age of 12. At 6 there developed a spastic condition of the extremities with choreo-athetoid movements. At 10 she became much worse and there were involuntary movements of the face and throat muscles which impeded speech. Only in the chewing movements was there occasional rest. Light chloroform anesthesia stopped these movements. Death occurred in severe coma. The brain was peculiarly dark and there was bilateral atrophy of the striatum and pallidum. The cortex appeared to be intact. With the Weigert stains the basal ganglions showed: "numerous sclerotic patches with many new vessels in the region of the globus pallidus, while the putamen and caudate were intact."

HISTORY OF CASES

Gallus' Case, Gerhard F.—A 7 months child, born in a difficult labor, who at the age of 6 months began to have convulsions, with ensuing rigidity and athetotic movements of the entire body musculature. He developed the ability to speak, but only indistinctly, though he could neither walk, stand nor sit. Pseudo-Babinski signs were present. Foerster's resection of the second to fifth lumbar and first and second sacral roots on the right side was done, and as a result the right leg could be moved. Psychic and body stimuli increased the spasm and the athetotic movements. In the last years of life there was an increase of spasticity with complete contracture of the legs and hips. There were severe involuntary movements of the face and upper extremities. Intelligence was limited. The facial expression was "timid." Death occurred at age 10.

Anatomic Findings: In a generally small brain, poor in medullary fiber, the putamen was abnormally wide and arched outward; the claustrum was also wide. On the other hand, the pallidum (especially *Gil*, Fig. 1), the corpus Luysi and in less degree the thalamus (*vtl, vtm and mv*) were small. The most oral striopallidal fibers in great part were missing. Further, there was no doubt of decrease in the thick striopetal fibers, at least in the inner half of the putamen. Most of the thick fibers that run between *Ge* and the thalamus and hypothalamus were missing. The thick fibers that connect *Gi* with the thalamus and hypothalamus were even more deficient. H^2 showed also a reduction to half and a large part of the pallidoluysian fibers had disappeared. The corpus Luysi was shrunken to about half, but in its remaining part contained practically normal fibers. In *vtl* the typical thick fiber bundles were not altered; *vtm* and *mv* were fiber-poor. Fiber disappearance in the substantia nigra, the nucleus ruber and its capsule could not be determined.

Comment: The authors consider this case of athetosis that began after birth a progressive disease of the striatum. They found principally a decrease of the fibers of the pallidum, as in the cases of Fischer and Rothmann which began later in life.

Thomalla's Case, Oskar M.—This was an eight months child, born in difficult labor, but not asphyxiated, who died at the age of 13 years. Slow constant movements in the throat and extremities began at the age of 1 month and soon involved the entire body; they were associated with spasticity which did not disappear in sleep. The spasticity increased under the influence of voluntary

movement and psychic stimuli. The left side was more affected than the right. There then developed disturbance of speech and, toward the end, dysphagia and retention of urine. With the general rigidity there was increase of reflexes and bilateral Babinski sign. There were no paralyses nor sensory disturbances and no intelligence defect. Anatomically, the cortex was normal. The striatum was smaller than in the preceding case. The pallidum, especially *Gi*, was very much reduced as in the former case. At the inner part of the pallidum it had led to severe fiber loss. This was located differently from that in the previous case. The oral striopallidal fibers were intact. The head of the caudate as well as the dorsal and ventral part of its middle region and the caudal part of the putamen were reduced. This condition was greater on the right. The lamellae of the pallidum and H^3 were richer in medullated fibers than in the previous case. The thick fibers of *vtl* were reduced. *Mv* and the commissura mollis were poor in medullated fibers. The corpus Luysi was reduced to about one-fourth, but stained well. On the other hand there was no abnormality of the nucleus ruber, its capsule or of the substantia nigra. The entire ganglions of the right side were less intensely stained than on the left.

General Comments.—The cause of the degeneration of the neurons in état dysmyélinique has remained obscure. Jelgersma has described as a "system disease" a similar anatomopathologic picture which will be taken up under the term état de désintégration. It should be noted that it is not the dying out of a single neuron system but a partial and varied consecutive degeneration. Part of the striopallidal, the thalamopallidal, the pallidoluysian and of the thick fibers going into *vtl* from H^1 are degenerated. The termination of the last named fibers, previously unknown, has been worked out by C. Vogt, in her myogenetic pictures. the fibers going from H^1 through H^3 into the pallidum. Accordingly they conclude, contrary to Jelgersma, that primary injury of the pallidum has the effect of killing all neurons that have a clear connection with the pallidum.

TOTAL NECROSIS OF THE STRIATUM

Thomalla's Case, Alfred L.—Clinically, this was a case of torsion spasm beginning nine months before death. In the last years of life there was a generalized muscular rigidity of the entire body. This patient became ill at the age of 12½ years with torsion movements of the right leg, and later of the hips and right arm. Four months after the onset speech was affected; two months later there was difficulty in swallowing and saliva drooled from the half-opened mouth. When admitted to the hospital, the patient showed rotary movements of the entire body with the exception of the left arm. Emotion increased the symptoms; active movements of the parts decreased it. Speech, after several words, was impossible. Sleep decreased the movements. Motor power, sensation and intelligence were intact. The knee jerks and skin reflexes were decreased. Babinski's sign was questionable; there was no beard; the genitals were underdeveloped. Once, under scopolamin, he showed athetotic movements of the right arm and bilateral Babinski sign. Death occurred from aspiration pneumonia. At necropsy there was found pneumonia, a colloid goiter and a small and nodular liver. Grossly the brain showed bilateral atrophy of the putamen with softer consistency than the neighboring tissue. Microscopically, no gross myelo-architectural changes were present in the cortex cerebri, only a general medullary poverty. (It could not, however, be determined how far the technical difficulties played a part.) The cyto-architecture of the motor area showed an abnormal retention of the embryonal

fourth layer. The pathology was centered in the striate system. In the left hemisphere there was a slight decrease of the caudate nucleus. The much atrophied putamen showed total necrosis of cells and fibers as described by Wilson in his cases. This had spread laterad not only toward the external capsule and even to the claustrum, but also mediad into part of *Ge*. *Gi* showed a fiber reduction. The corpus Luysi was not only smaller, but also fiber-poor; the rest of the hypothalamus, as well as the thalamus, especially the outer half of the region of *va* and the peduncle showed no anomalies. The right hemisphere showed similar changes except that the necrotic process was located in the oral part of the putamen and its neighboring part, and was less severe in the caudal portion. In the thalamus, in the region of *va*, there was moderate loss of fibers as on the left side. The liver showed cirrhosis identical with that described by Wilson.

Comments: The disease picture was that of a torsion spasm. That, at the bottom, there was a progressive disease of the striate system was evident. The authors were, however, astonished to find a pathologic process which in nature and location, including the liver cirrhosis, was like that of Wilson's disease. The authors feel it is not yet possible to state the cause of the great differences in the clinical pictures. In the foregoing case the putamen was the primary site of the disease which had spread to the neighboring structures. The bilateral fiber poverty in the region of the thalamic nucleus *va* needs explanation. Whether the relatively long freedom of the left arm is related with the slightness of the disease in the caudal part of the right putamen must be determined; it will assist toward a finer somatic localization, not only in the putamen, but also in the pallidum. The contrast between the severe degeneration of the corpus Luysi and the integrity of the rest of the hypothalamic region and that part of the thalamus which was hitherto brought into relation with the striate system was striking. The conclusion must be that *Ge* sends relatively few fibers to the striate body. The better preservation of the medullated fibers in the oral part of the left corpus Luysi speaks for a relation between its fibers and the caput caudati and the most oral part of the pallidum.

LOCALIZED NEUROGLIA LESIONS IN THE STRIATUM WITH CONCOMITANT PRESENILE CHANGES OF THE STRIATE SYSTEM

Westphal's Case, Johann Reichardt.—At 43 the patient began to have athetoid movements in the proximal joints of the left leg and arm, and also in the face and in the tongue. After several weeks the same sort of movements began in the rest of the body. There resulted then, a general athetosis which was increased by psychic stimuli and a torsion spasm which varied in its intensity. It was not increased by passive movements, but was lessened by active movements, with increasing dysphagia, dysarthria, salivation, and brachybasia. The body was bent over, with retropulsion and lateropulsion, mask-like face, slow movements, and urinary dribbling. The abdominal reflexes were present and the knee jerks undisturbed. Memory was impaired, especially perceptive memory. Seven weeks after the onset of the disease diarrhea set in and in three weeks the patient died. Anatomically, there was perivascular cell infiltration in single vessels of the cortical white matter. Westphal and Sioli found chronic changes and true degeneration in the cortex of the first frontal convolution and in a lesser degree in the anterior and posterior central convolutions. The area gigantopyramidalis showed no

architectural change. The striatum and pallidum contained numerous vessels with perivascular infiltration. The cells consisted mainly of lymphocytes with some plasma and mast cells. The tunica media of many blood vessels in these areas showed hyalinization. The capillaries of certain areas of the pallidum were covered with lime salts, which hitherto has been recorded only in one case of paralysis agitans, and by Walbum and Catola as an accidental finding in tabes. The above pathologic findings had not led to parenchymatous destruction. Yet as the result of loss of parenchyma, an état criblè resulted, especially in the striatum and neighboring parts. The most essential pathologic change consisted of numerous patches in which the normal tissue was replaced by neuroglia tissue. This was present especially in the striatum. The etiology of the process is unknown.

Comments: This was a peculiar case in which the patient showed at the beginning a localized athetosis which soon became generalized; it suggested torsion spasm and paralysis agitans sine agitatione. Death occurred in ten weeks from an intercurrent disease. The microscopic findings although observed in other parts of the brain were especially manifest in the striatum and pallidum; there were presenile changes with marked parenchymatous atrophy that was confined to striatum and pallidum but were more particularly present in the striatum.

ÉTAT DE DÉSINTÉGRATION

C. Vogt investigated cases of paralysis agitans and in all, whether with tremors or without, found pathologic changes constantly in the striatum and pallidum. These changes she has called état de désintégration in close agreement with Marie. She also examined normal brains and in no case without paralysis agitans did she find similar changes. Moreover, in two cases whose clinical history was unknown, she made a diagnosis of paralysis agitans on the basis of the pathologic anatomy, a diagnosis that was afterward substantiated. The Vogts in their former work have discussed the meaning of the term état de désintégration in various diseases which are associated with degeneration of the striatal and pallidal tissue: (1) Those in which volume reduction was caused by disappearance of the ganglion cells and fibers as an état paradysmyélinique and which approach the picture of an état dysmyélinique; (2) small areas of softening from necrobiosis or hemorrhage with the production of cavities (état lacunaire); (3) rarefaction and eventual resorption of the tissue and blood vessels. This necrobiotic process begins with lightly stained areas which can be recognized with the van Gieson stain. An atrophy of the ganglion cells follows and the glia nuclei increase in number. The nerve fibers then disappear. Connective tissue elements and new blood vessels are formed later.

In the smallest blood vessels there is a tendency to the condition described by Durand-Fardel, in 1854, as état criblé. The resorption of tissue around the larger blood vessels leads to the état lacunaire of Marie. This can be designated as état prélacunaire. The Vogts recall the fact that such parivascular extensions occur not only around arteries but also around veins. As P. Marie has noted, the process around larger blood vessels is not different from that around the smaller ones. The etiology is different from that in the lacunar form mentioned under (2). The Vogts subdivide as follows: all distended perivascular lymph spaces are called "criblé"; the adjoining portions are called "précriblé"; and the title "lacunaire" is reserved for the processes mentioned under (2).

REPORT OF CASES

Etat de désintégration may occur with or without severe arteriosclerotic or senile dementia.

(1) *Without Severe Dementia:* The following cases show clinical manifestations similar to those of paralysis agitans:

Bielschowsky's Case, Karoline V.: Clinically, this was a typical case of paralysis agitans with violent tremor. Anatomically, sections revealed dilatation of the ventricles. There were no changes belonging to a senile or arteriosclerotic dementia. Etat criblé was present in many parts of the brain, but especially in the striatum. These changes in the striatum and pallidum were so recent that they were thought to have no bearing on the clinical manifestations. The caudate nucleus was atrophied.

The essential pathologic change, in addition to atrophy of the caudate, was an état criblé in various parts of the brain, especially in the striatum. It is interesting to note that with the disease most marked in the striatum there was severe tremor.

Bielschowsky's Case, Wilhelmine P.: A case of paralysis agitans without tremor. Anatomically, there was found in the white matter of many convolutions an état criblé which had not led to degeneration of any known fiber system. The caudate nuclei were evidently atrophied, especially the left. It showed, as did the putamen, a moderate état criblé; large vessels were found in the putamen. Finally there was an especially intense état criblé in the pallidum, mainly in the anterior part. Aside from this, there was a fiber lack (a partial état paradysmyélinique) which suggested cases of état dysmyélinique. The corpus Luysi was slightly atrophied.

Comment: Here the pathologic changes in the striate system were of the type of an état de désintégration. In contrast to the former case (with tremors), the pallidum was more involved than the striatum. This fact bespeaks the evident atrophy of the corpus Luysi. Clinically, the case was distinguished by the fact that there were no tremors, but instead an intense rigidity which the authors in their former work have designated as the "pallidal syndrome." The fact that such severe disease existed in the pallidum agrees with the theories of the authors. The contrast between the normal H^2 bundle and the atrophic corpus Luysi should be noted.

Freund's Case, Pauline H.: A case of paralysis agitans of two years' duration which showed rigidity and only slight tremors, probably limited to the left hand. The rigidity was especially marked in the upper extremities and particularly in the left. Anatomically, there was a form change in the striatum and an état criblé in the putamen, more marked on the right than on the left. The pallidum showed severe volume reduction, more in the right than the left. In the pallidum, especially on the right, many of the thicker fibers were degenerated. H^2 showed a slight volume reduction, the right more than the left. The corpus Luysi was much reduced, again more on the right than the left.

Comment: There was no doubt that the main pathologic change was in the striatum system. In addition to an état criblé in the putamen there was partial disappearance of the thick fibers of the pallidum as an état paradysmyélinique. The cause of the localization of the lesion is explained as in the case of état dysmyélinique. Because rigidity was prominent, it was theoretically presumed that pallidal disease existed. This was confirmed. Of the arms the left only showed rigidity; the left leg was more rigid than the right. This led to the assumption that the lesion was in the oral part

of the right pallidum. This was verified. The theoretical relation of tremor to the striatum was verified by finding an état criblé especially in the right putamen. Finally the état paradysmyélinique of the pallidum and the severe atrophy of the corpus Luysi were parallel and this was more severe on the right side. A true decrease in the size of H^2 and H^1 supports the cases described under état dysmyélinique.

Marie's Case, Renoult: A hitherto well man, aged 72, showed typical paralysis agitans with tremors of the left hand two or three days after a severe fall. Tremors soon appeared in the right hand, then in the left foot, and finally in the right. Rigidity remained slight; there were no pseudobulbar symptoms. In the left hand there was ankylosis of the joints probably of rheumatic origin. Pathologically, there was a large area of softening in the caudal part of the medial side of the left hemisphere, with a widespread état criblé, which was especially marked in the white matter of the island of Reil and in the striatum.

Comment: There was here, in an état criblé of the striatum, one of the forms of état de désintégration. It was remarkable that the pallidum and the pallidal system were relatively intact. The particular localization in the striatum indicates, according to the Vogts, predominance of tremors with slight rigidity in the clinical picture.

Freund's Case, Bertha Z.: A typical case of paralysis agitans with tremor and rigidity, the patient dying at the age of 82. Pathologically, there were senile changes in the cortex. The caudate was atrophied, with beginning état criblé in the putamen and especially marked fiber loss in the outer two-thirds of the left external lamella and an even greater loss in the outer half of the left internal lamella in the region of the hypothetical arm zone. The authors conclude that this case adds justification to their hypothesis of pallidal and striatal localization.

Schuster's Case, Marie B.: A case of paralysis agitans in a man who died at the age of 62, after having shown marked rigidity with propulsion and retropulsion. Pathologically, besides smallness of the striatum and pallidum, and an état criblé in these structures, there was also partial disappearance of the fibers of the pallidum especially in the most caudal part.

Comment: Paralleling a marked rigidity there was a severe pallidal disease marked in the caudal area where is located by the authors the center for the back and legs. Propulsions were most prominent in this case.

Lemos' Case, J. D.: This is one of the two cases in which C. Vogt made a diagnosis on the basis of the pathologic findings. It was diagnosed by Lemos as paralysis agitans with pseudobulbar palsy and was reported as such in Anales scientificos de Faculdade de Medicina do Pôrto in 1912. In brief the disease, beginning at 17, was a paranoid condition accompanied by symptoms of paralysis agitans sine agitatione with bulbar palsy. Pathologically, there was a severe lacunaire condition in the sense of Marie, with numerous small areas of softening and isolated areas of hemorrhage. There was marked état criblé in the white matter of the hemisphere and in the central ganglions; also numerous changes in the pons and in the white matter of the cerebellum. This état lacunaire et criblé had probably occurred in the last year of life. The striatum, pallidum and thalamus were small and part of the striopallidal fibers were degenerated.

Comment: It was because of the état de désintégration that C. Vogt made the diagnosis of paralysis agitan. The severity of the changes in the striatum

and pallidum, and the tendency to propulsion and rigidity were not clear. The bulbar symptoms were explained due to disease of the head of the caudate and disappearance of part of the fibers to the pallidum. The disproportion between the mild caudate disease and the very severe bulbar symptoms became clear from the severe lacunaire lesions in the white matter of the cortex and in the thalamus. The authors attribute the severity of the striate symptoms to the concomitant disease of the cortex.

Lemos' Case of a Negro, Aged 100: The brain of this man was given to C. Vogt as a normal brain and she was astonished to find in the striatum and pallidum a marked état de désintégration. When the history was obtained it was found that there had been symptoms of paralysis agitans. Pathologically, aside from a diffuse atrophy of the medullated fibers of the cortex and a few areas of softening in the cortex and thalamus, there was a marked état criblé in the striatum and pallidum. There were none in the album insulae.

Comment: The fact that C. Vogt made the diagnosis on the basis of the état de désintégration is interesting. The caput caudati and its fibers were not nearly so severely diseased as in the former case. No pseudobulbar symptoms were present and the authors feel that bulbar symptoms make severe lacunaire disease of the white matter of the hemispheres, especially of the thalamus, probable. The rigidity must have been due in part to the medullated fiber atrophy of the hemisphere. Etat criblé in the putamen without involvement of the insula is of interest.

Westphal's Case, P. Grohe: A syphilitic and alcoholic man, aged 65, with Argyll Robertson pupils, six months before death developed paralysis agitans symptoms without tremor. Pathologically, besides numerous large areas of necrobiosis in the rest of the brain, severe changes of the same kind were found in the striatum and pallidum.

Comment: This was a case of paralysis agitans very late in life. There was a "lacunaire" condition in the striatum and pallidum, to which is attributed the paralysis agitans syndrome.

(2) *With Severe Senile Dementia:*

Reich's Case, Hermann W.: A patient, aged 65, with senile dementia on an arteriosclerotic basis, developed progressive muscular rigidity, greater on the left side and more in the arms and hips. There were slight static tremors of the hands, and facial rigidity. Pathologically, the cortex showed, especially in the frontal pole, a severe cell decrease in the various layers. In the motor area no such change was found. The cortex elsewhere contained numerous "drusen." The album gyrorum showed a moderate état criblé. The album centrale, the capsula interna, the pes pedunculi, thalamus, nucleus ruber, cerebellum, pons and medulla were without severe pathologic change. On the other hand the striatum, and in less degree the pallidum and corpus Luysi, were atrophied. In the striatum a moderate amount of état criblé was present.

Comment: In this case the diagnosis was made from the pathologic picture without knowledge of the clinical history. Not only were the authors able to form a judgment of the severity of the clinical manifestations, but they believe that the changes in the striate system were sufficient to account for the severe rigidity. Of importance for elucidation of the fiber connection is the fact that with severe atrophy of the fibers in the striatum, the fibers running between the striatum and pallidum were well preserved. These fibers are the axis cylinders of the interposed cells of the striatum. The cause of the severe dementia is found in the cell disappearance of the frontal lobe as contrasted with the integrity of the cytoarchitecture of the motor area. Through-

out the cortex were numerous "drusen," or spaces recognizable by their granular background. They had definite relation to the blood vessels and contained many glia nuclei.

The origin of the état criblé (miliary degeneration) is important. Bielschowsky thinks that these result from a hindrance to the outflow of lymph as a result of stoppage of the adventitial lymph space. He supports this view by the demonstrable fibrosis of the adventitia of the vessels. Marie and his students believe that it is due to the "corrosive action" of material from the arterial walls and they speak of a "vaginalitis destructiva." This material must also come through the veins since the same condition is seen around them. At present all this is theoretical.

GENERAL COMMENT

All the cases cited can be classified as paralysis agitans even though complicated with other cerebral disease.

(a) *Symptomatology.*—The disease may begin with a monotremor and then spread; or it may run its course without tremor. The rigidity then comes to the fore; it is increased by peripheral stimuli and emotion, and is decreased by active and passive movements. Malaisé has come to the conclusion that brachybasia is due to incoordination with slight paresis, and that rigidity may coexist without relation to it. He has therefore confirmed the views of Bielschowsky, Maillard, and Zingerle. Foerster has recently substantiated his findings.

(b) *Pathophysiology of Paralysis Agitans.*—It must first be stated that the entire symptomatology of paralysis agitans can remain constant. The Vogts do not attribute the symptoms to irritation from a necrobiotic process, but rather to absence of function. This theory has the following basis: The disease begins with the tremor. The fact that Wilson's disease may begin with a similar tremor demonstrates that tremor may be a symptom of the "syndrome of the striatum." That paralysis agitans may begin similarly can be deduced from the fact that in two cases of paralysis agitans (Bielschowsky's case of Karoline V. and Marie's case of Renoult) the pathologic change was most intense in the striatum. The tremor manifests itself here as a "pallidal hyperkinesis." The question arises as to why état de désintégration produces tremor, and état fibreux and gross lesions produce choreic movements. Etat de désintégration is a much slighter process than the other two diseases and injures the function of only a part of the striatal cells. The authors point out that in Wilson's disease, with increasing involvement of the striatum, there is produced not tremor nor involuntary movements but a rigidity which prohibits movement. Previous involvement of the pallidum leads to rigidity without tremor as in the case of von Economo. The Vogts believe it is permissible to formulate the present day hypothesis that in paralysis agitans sine agitatione there has been previous disease of the pallidum which has produced rigidity and thus prevents tremor. The increase in the tremors in paralysis agitans by peripheral stimuli and psychic influences, becomes intelligible when the connections of the pallidum with the periphery and the cortex are considered. Rigor is a pallidal symptom and is attributed to severe inhibition of pallidal function. This is supported by Bielschowsky's case Wilhelmine P. In Lemos' case J. D. and Reich's case Hermann W. there was an increase of rigidity by injury of the prefrontal "denervation" function. The increase of rigor by peripheral stimuli and especially by psychic stimuli becomes intelligible by analogy with tremor.

Various and diverse changes have been included under the term état de désintégration. In one case many forms may be combined. The authors believe that paralysis agitans is not the result of primary alteration of the entire cerebrum by senility, but that the striate system, especially the striatum and pallidum, tend themselves to produce presenile disease. They do not subscribe to syphilis as a factor, and the work of Camp is quoted to substantiate this. Is it possible to make the diagnosis as to what type of état de désintégration is present in a given clinical case? According to present indications an état lacunaire as the result of état de désintégration is to be expected when: (1) The disease advances very acutely (Westphal's case P. Grohe); (2) severe bulbar symptoms are present with other slighter manifestations (Lemos' case J. D.); (3) slight strokes occur with only few symptoms of paralysis agitans, and with more or less pronounced pseudobulbar symptoms.

Progressive tremor with slight rigidity speaks for the possibility of an état criblé in the striatum. Outspoken rigidity occurred with disease of the pallidum. With very slow onset of tremor there was usually either an état criblé of the pallidum as in Lemos' case of the negro aged 100, or a combination of état criblé and état paradysmyélinique. The material gave no clue to the diagnosis of these two conditions during life. The Vogts believe that serology may help to solve the problem. Sicard could find neither chemical nor cytologic modification in the cerebrospinal fluid in paralysis agitans, but in lacunaire and pseudobulbar cases he found increase in the albumin.

GROSS LESIONS IN THE STRIATE SYSTEM

Liepmann's Case, Elisabeth L.—At 43, the patient began to have an hallucinatory confusion with dementia and soon showed confused and inarticulate speech. In her sixty-seventh year, choreic movements began in the right arm and increased in violence. After another stroke, there was paresis of the right corner of the mouth and the right arm with a cessation of the choreic movements for twenty-four hours. They began again and in the right leg in addition, but without paralysis. The movements persisted until death at the age of 69. The knee jerks were much increased, the right more than the left. Doubtful Babinski signs were present. Hypertonia occurred in the legs in the last year. One month before death a slight left sided hemiplegia occurred. Pathologically, there was degeneration of the left caudate nucleus, the anterior part of the internal capsule and the oral half of the putamen. The choreic movements were considered to be due to the left striatal lesion.

CONCLUSIONS

The Vogts have described thirty-three cases of striate system disease and draw the following conclusions: (1) The striate system as they delimit it includes various functions. Destruction of gray matter of higher or lower physiologic level produces finer or coarser symptoms. Disturbance of finer striatal function leads to involuntary movements; while coarser pallidal function disturbances shows grosser movement defect accompaned by rigidity. In this way are explained the dissimilar clinical pictures from lesions in the same region; (2) in the various diseases, the inhibition of pallidal function may dominate the picture; (3) in the cases studied the strict striatal system has controlled only the opposite side of the body; (4) no cases have yet been described of a similar symptomatology in which the striate system, in the limited sense, was intact.

THE STRIATE SYNDROME

The authors distinguish between irritation and loss of function. The loss of function syndrome is limited to état marbré, état fibreux, état de désintégration and gross lesions. The characteristic symptoms are: (*A*) Striatal akinesis, which is a component of poverty of mimic expression as well as of associated movements, changes of position, movements of orientation, protection, and defense deflexes; (*B*) incoordination, especially in the bulbar muscles, in walking and standing; (*C*) substriatal (pallidal) hyperkinesis: (1) involuntary movements (athetosis, choreic movements, etc.) which can be suppressed for a moment, and can be called forth or exaggerated by peripheral stimuli, particularly psychic stimuli of an agreeable nature; (2) hypertonic conditions: (a) The duration and intensity are not known since pure striatal disease was not present in their cases; (b) they are increased by peripheral stimuli, especially emotional stimuli, but are not exaggerated by continued irritation; they are decreased by nonirritative active and passive movements; (c) certain muscle groups or affect agonists and antagonists are attacked equally; (d) they produce a true diminution of muscle power or a slowness of motion; (e) perhaps a pathophysiologic hypertonia which is as yet not clear; (f) the absence of other disturbances. Such a picture is not produced by any other portion of the brain.

The interpretation of the normal physiology is due to five anatomic facts: (1) The striatum and pallidum of man show neither "evolution" nor "devolution" as contrasted with the same organs in the cercopithecine brain. The same functions must therefore be attributed to both man and the lower apes; (2) the fibers of the pallidum become myelinated so much earlier than the cortex or striatum that a time existed when the highest motor functions were carried on by the pallidum; (3) in the corticospinal and the corticopontocerebellar tracts, corticifugal fibers are present, which, avoiding the pallidum and striatum, can affect the peripheral motor neurons; (4) the pallidofugal and striofugal fibers originate, at least in man, from the thalamus and its neighboring gray nuclei; (5) the subpallidal tracts do not act directly on the peripheral motor neurons.

WINKELMAN, Philadelphia.

THE ETIOLOGY AND PATHOLOGY OF SCIATICA. KARL PETREN and EDITH OTTERSTRÖM, Act. med. Scand. **55**:614, 1921.

There have been many theories as to the site of the lesion producing the disease known as sciatica. Some authors have claimed that it is in the sciatic nerve itself, some that it is in the posterior nerve roots and some have even suggested its presence in the muscles of the lower extremities. This article is chiefly concerned with the possible relationship of spondylitis deformans with sciatica, and the condition of the spinal fluid in patients suffering from this disease.

In eighty cases of sciatica, roentgenologic studies were made of the vertebral column. In forty-five it appeared normal, in twenty-five there were some very slight arthritic changes, just enough to permit a diagnosis of spondylitis deformans. In the remaining ten cases there was a much greater degree of osseous and joint change, constituting them well pronounced cases of this disease. The question, of course, arises as to how many people of advanced or middle age, who do not have sciatica, would show changes in the vertebral column. They quote Kahlmeter as having found pathologic changes

in 17 per cent. of patients complaining of symptoms other than those referable to the spinal column or nerves. The authors conclude, therefore, that the patients suffering from sciatica who have changes in the vertebral column are relatively few in number and the arthritis is not sufficiently important in itself to play much part in the causation of sciatica. If there is any etiologic relationship between the two affections, it is necessary also to admit the existence of some other factor to explain the fleeting and temporary character of the symptoms of sciatica and the relatively steady progression and fixed character of the alterations produced by spondylitis deformans.

In forty-nine patients lumbar puncture was performed. In forty-seven of these the fluid was normal, although in twelve there was a slight increase of pressure. The Nonne reaction was negative in all these fluids, except those that had been contaminated by blood, and the cell count was always below 4 cells per cubic millimeter. The Wassermann reaction was negative in the spinal fluid of all the patients. Of the two cases in which the spinal fluid showed pathologic change, the first case is given in detail. It concerned a man, aged 60, with a history of pain in the back for ten years and a sciatica of three months' duration. Laségue's sign and tenderness along the sciatic nerve were present on both sides. Lumbar puncture in his case revealed: a pressure of 170 mm. in the spinal fluid, a positive Nonne test, no lymphocytes, and a negative Wassermann reaction. The puncture was repeated a month later and showed the same changes. However, he left the hospital a few days later apparently relieved of pain. It was considered that his trouble was meningitic rather than a true sciatica, and the authors were unwilling to deny the possibility of his having syphilis.

The other patient was a man of 23, with a two weeks' history of severe pain in the back and in the posterior aspect of both lower extremities, worse at night-time and less marked during the day. He had a bilateral Laségue sign, tenderness along both sciatic nerves, and walked with his back held rigid. Lumbar puncture showed a pressure of 205 mm. and 28 lymphocytes per cubic millimeter. The Nonne and Wassermann reactions were both negative. The possibility of a syphilitic meningitis irritating the posterior roots, was considered, but no other evidence of syphilis was discovered. He was treated, however, with mercury inunctions, iodid of potassium, and acetyl-salicylic acid, and was kept in bed. Lumbar puncture performed three weeks later showed a pressure of 22 mm. with 24 cells and a strongly positive Nonne reaction. At the end of his course of mercurial inunctions he was given a third lumbar puncture. The pressure at this time was 160 mm., there were 5 cells and a faintly positive Nonne reaction. The patient was discharged forty-seven days after his admission, apparently well. The final diagnosis was syphilitic spinal meningitis. The authors quote these two cases as of importance in the consideration of a possibility of syphilis giving symptoms like sciatica. In most of the quotations from other writers, there is an apparent agreement on the relative rarity of changes in the spinal fluid. Dejerine, however, who promoted the theory of sciatica as a lesion of the roots rather than of the nerve itself, has published a case report with a marked lymphocytosis in the spinal fluid. This patient, however, had paralysis of certain muscles of the leg, which would not entitle the case to classification in the category of sciatica. Those patients whose spinal fluids showed a lymphocytosis cannot escape the suspicion of having syphilis and, in that case, would

not be true cases of sciatica according to the authors. However, Dejerine seemed to consider that a large number of cases of sciatica were due to irritation of the posterior roots in syphilitic meningitis. If this conception be true, it is evident that the percentage of cases of sciatica should be in proportion to the frequency of syphilis in any given population; and this is certainly more or less in accord with the diffusion of syphilis in different populations. Be that as it may, the cases coming under this heading could only be those in which either there were changes in the spinal fluid or sensory and motor disturbance in the lower limbs. The authors consider those cases with positive spinal fluids and other changes as coming under the heading of secondary sciatica rather than the more common types of sciatica which do not show such changes. There seems to be nothing in the literature to show that spinal fluid changes in sciatica are at all common and the presence of such changes excludes an ordinary sciatica.

PARKER, Rochester, Minn.

FURTHER OBSERVATIONS ON THE ROENTGEN-RAY TREATMENT OF TOXIC GOITER. J. H. MEANS and G. W. HOLMES, Arch. Int. Med. **31**:303 (March) 1923.

In this paper, which covers observations extending over more than three years and a prewar period of two years, Means and Holmes discuss the value and limitations of roentgen-ray treatment of exophthalmic goiter and toxic adenoma. It is suggested by the authors that forthcoming papers by their colleagues giving the results of treatment by surgery alone and by surgery and roentgen ray combined be read in conjunction with the present communication.

After reviewing the history of roentgen-ray treatment of toxic goiter and describing the methods of their clinic, the authors present the data of their study. One group was made up of recent cases, another of prewar cases. There were fifty-eight cases in the recent series, of which forty-four were cases of exophthalmic goiter and fourteen were cases of toxic adenoma. "Of the forty-four exophthalmic goiter cases, sixteen showed little or no improvement; eight of these came to operation later and were apparently cured. Of the twenty-eight cases in which there was improvement, twelve were apparently cured and sixteen were improved but were not rendered entirely free from hyperthroyidism. No cases seemed to be made worse by the treatment." A composite chart of the twenty-eight cases showing improvement indicates "a rapid fall in pulse (from 115 to 89) and basal metabolism (from + 55 to +21 per cent.) and a corresponding gain in weight (9 per cent.) in the first four months of treatment, during which time the patients received, on the average, five treatments. No further significant drop in pulse or metabolism curves occurred in the next six months, in spite of more treatment." All the toxic adenoma cases showed improvement and five were apparently cured. The curves of the toxic adenoma group show a more gradual decrease in pulse and metabolism and a more gradual increase in weight. In this group as a whole a normal pulse and metabolism level is finally reached. Of the prewar group the authors were able to examine nine patients: six have normal basal metabolic rates five to six years after the first treatment; one has a slight elevation, + 15 per cent., but no clinical symptoms; two patients still had elevated metabolism and clincally were better though not well.

The authors recommend roentgen-ray treatment of exophthalmic goiter for four or five months; if the patients do not respond or still remain somewhat

thyrotoxic after this period, surgery should be considered. In toxic adenoma the thyroid appears to be the actual seat of the disease and here the authors believe the indication for surgery is more definite; but roentgen-ray treatment is to be used when operation has been refused or when the patient is too thyrotoxic for a safe operation.

VONDERAHE, Philadelphia.

DYSTROPHIA MYOTONICA (MYOTONIA ATROPHICA). W. J. ADIE and J. G. GREENFIELD, Brain **46**:73 (Part 1) 1923.

In presenting this subject the authors review 123 cases in the literature and report four cases of their own, one with necropsy. The general characteristic features of the disease are myotonia, muscular atrophy and general trophic disturbances. Dystrophia myotonica is considered to be heredofamilial. The fully developed disease is confined in the main to one generation; while ancestors do not suffer as a rule from myotonia or muscular atrophy, they often have presenile cataract or other degenerative phenomena. The age of onset of the disease is usually between 20 and 35 years.

Myotonia is one of the chief symptoms and is usually best observed in the hand grasps, but is also expressed in "buckling up" of the feet on beginning to run, stiffness in the knees after squatting, and stiffness in the jaws during mastication; sudden falling, as in Thomsen's disease is another manifestation regarded by the writers as myotonia, as is also the marked incoordination seen in rapid extension and flexion of the limbs. In contrast to this active myotonia, mechanical myotonia with persistence of contractions produced by percussion of the muscles was more widespread. The classical myotonic reaction to electrical excitation was seldom found, but some minor modification was usually present. The authors maintain that myotonia is always present in some stage of the disease and that this symptom is myogenic in origin.

Muscular atrophy is said to be a common finding, and the following regions are nearly always attacked: the facial muscles, the muscles of mastication, the sterno-mastoids, the forearms, the vasti of the thighs or dorsiflexors of the feet; it is usually present in more than one of these groups when the patient first comes under observation, but it may affect any one of them first, or most, or alone. From the regions first affected the atrophy may spread to other areas. The authors do not believe that pseudohypertrophy accompanies this disease, and maintain that fibrillary tremors are never present. Except for its selection of the extremities of the limbs, they call attention to the close similarity, both clinical and pathologic, of the atrophy of dystrophia myotonica to that of the progressive dystrophies.

Dystrophic symptoms which make up the third group of the symptom complex of this disease are: cataract, atrophy of the testes and loss of sexual power, baldness, general wasting of the body tissues, cyanosis and coldness of the extremities.

Necropsies have been performed in six cases of this disease including one by the writers; in none were there any pathologic findings in the cord. In the authors' case there was an increase of colloid in the pars anterior and pars intermedia of the pituitary body; in the suprarenal glands there was a patchy distribution of the cortical lipoids. There were striking changes in the muscle tissue examined at biopsy and at necropsy. In the early and middle stages of the disease there was disproportion in the size of the muscle fibers, with a large increase of the sarcolemma nuclei which invade the fiber itself

and form long chains down its center. The writers regard this picture as a characteristic feature of dystrophia myotonica. In the later stages the muscle fibers disintegrate and are replaced by fat and connective tissue. The authors do not accept the claims of some that this disorder is due to a disturbance of the endocrine glands.

STACK, Philadelphia.

THE SIGNIFICANCE OF THE FRONTAL LOBES FOR THE HIGHER PSYCHIC FUNCTIONS. JULIUS DONATH, Deutsch. Ztschr. f. Nervenheilk. **76**:281 (March) 1923.

The author reviews the literature on this subject. According to Camper's facial angle (index for intellect) the Caucasian race measures from 80 to 85 per cent., the negro 70 per cent., the young orang 76 per cent. Some neurologists (Monakow, Monk and others) deny or attach slight importance to the psychic significance of the frontal lobes. They base their views on the experimental work on animals of Fritsch and Hitzig in 1870, of Ferrier in 1873, and of Beevor and Horsley in 1887. The author believes that little, if any, scientific data can be obtained from subjects of such low intelligence level. Clinical observations have definitely established the importance of the frontal lobes. Those who oppose this statement believe that motor functions only exist in these regions.

Definitely, there have been established: (1) Broca's speech center in the posterior third of the left third frontal convolution; (2) the tractus fronto-pontocerebellaris extending from the frontal lobe through the pons to the cerebellum. Its definite point of origin in the frontal lobe is undetermined; according to Flechsig in the first, to Dejerine in the third and to Brodmann in the first and second convolutions. Disturbances of this tract supposedly cause frontal ataxia. Similary Bárány in 1913, and Szasz and Podmaniczky in 1917, reported that spontaneous past pointing can occur with frontal lobe tumors; (3) projection fibers from the second and third frontal convolutions to the subcortical ganglions; (4) association fibers, fasciculus longitudinalis superior, connecting the second and third convolutions with the occipital and temporal lobes, and Monakow's fasciculus frontocentralis; (5) the graphic and motor musical centers, probably located in the left second frontal convolution.

In frontal brain tumors paresis of the neck muscles has been observed, also typical paralysis agitans syndromes. This latter syndrome is probably due to pressure on the corpus striatum. Other neighboorhood symptoms are jacksonian epilepsy, paralysis of the extremities, etc. Against the theory of the psychic functions of the frontal lobes, the author quotes from the literature a series of frontal brain tumors, unilateral and bilateral, and of extensive frontal brain injuries (thirty cases) without definite psychic disturbances. He explains this by citing the fact that brain tumors frequently run a symptomless course, or by the suggestion that the observations were not sufficiently accurate.

In favor of the location of psychic functions in the frontal lobes, Donath reports cases (tumors and injuries) under three headings: (a) simulating general paralysis (five cases), (b) schizophrenoid syndromes (nine cases), and (c) marked dementia (nine cases).

Donath further calls attention to the early and constant psychic disturbances that occur in tumors of the corpus callosum, probably as the result of the indirect involvement of both frontal lobes. According to Gaspero, mental changes, especially child-like dementia, occur most frequently in callosal tumors, next in frontal tumors; stupor is frequent in tumors of the frontal half of the

cerebrum. Melancholia, hallucinations and paranoia have no localizing import. In frontal brain lesions, impairment of memory, retrograde amnesia, confabulation, impairment of intelligence, euphoria, hypomania, irritability and outbursts of temper, uncontrollable laughter and crying, moral insanity, and undue drowsiness are most frequent.

HAMMES, St. Paul.

BRAIN TUMORS IN YOUNG CHILDREN. A CLINICAL AND PATHOLOGICAL STUDY. MARTHA WOLLSTEIN and FREDERIC H. BARTLETT, Am. J. Dis. Child. **25**:257 (April) 1923.

Wollstein and Bartlett find an incidence of 0.2 per cent. of brain tumors in 4.563 necropsies. The ages ranged from two weeks to three years, the average being 15 months; there were two girls and seven boys. Seven of the nine cases are analyzed in this paper. All of the tumors were gliomatous in type. There were two supratentorial tumors, one of which occupied the basal ganglions; the other ocupied the left cerebral hemisphere and occurred in a child two weeks old so that the congenital origin appears evident. There were five infratentorial tumors in all of which the vermis of the cerebellum was involved. The medulla and pons were infiltrated in two cases, and the right cerebellopontile peduncle was involved in two cases.

The physical signs were variable. The authors point out that in infancy there is a growing brain and a distensible skull so that manifestations of the neoplasm are delayed. Vomiting did not appear as a significant symptom in any case; convulsions were similarly inconspicuous in the symptomatology, occurring in only one case and in that just before death. The spinal fluid showed no constant changes; xanthochromia was present in only one case. The one constant manifestation was hydrocephalus.

VONDERAHE, Philadelphia.

THE PHENOMENON OF THE INDEX FINGER. J. M. RAIMISTE, Rev. neurol. **29**:387 (April) 1922.

The author speaks of two forms of this phenomenon: passive, and active. The first is best seen when the forearm is at rest in the prone position and the hand is hanging limply. The index finger hangs or extends in a plane beyond that assumed by the other three fingers, the distal phalanx projecting farthest beyond the plane of the other fingers, and the proximal phalanx least. The active form of the phenomenon of the index finger is best demonstrated by making a fist, or by actively extending all the fingers. The index finger then lies approximately, at all points, in the same planes as the other fingers. This phenomenon also appears to be a manifestation of certain emotional states: the passive, of sad, depressed, or indifferent moods; the active, of the more tense moods, such as anger, fear, disgust, etc.

The writer then discusses the conditions under which this phenomenon, active or passive, may be absent or modified.

Preliminary to a detailed discussion of the presence or absence of the phenomenon in central or peripheral palsies, the writer raises the question whether the phenomenon is not purely mechanical. Based on experiments on the cadaver, he concludes that it is mechanical, as the sign appears as distinctly in the cadaver before rigor mortis sets in as it does in the living subject. This is particularly true of the passive phenomenon. It is in cases of peripheral nerve palsies, particularly, that he assumes that the presence or absence of

the phenomenon is due to normal muscle tone; this is particularly true of the active phenomenon. In all cases of profound narcosis, when the tendon reflexes were absent and muscle tone was lacking, the passive index phenomenon was always manifest.

Then follows a discussion of particular cases in which central or peripheral nerve palsy was present. The paper concludes with the following summary:

A. In cases in which: (1) The individual having both arm and hand in the limp position, the phenomenon of the index is absent or is weakened; and (2) when, with this, there is no contracture of the fingers, it is logical to conclude that there is paresis of the extensor muscles of the index finger of peripheral character (e. g., an isolated paralysis of the radial nerve, as in polyneuritis).

B. (1) In pronounced organic paralysis of central origin, the patient, in opening the hand and fingers, still manifests the condition of a raised or elevated index finger; i. e., the active index phenomenon is preserved; (2) in two pronounced cases of functional paresis of the entire upper extremity, when the patients opened the fist the index finger remained in the same plane as the last three, that is, the phenomenon of the index finger was absent. Such a failure in the absence of contracture or spasticity speaks in favor of a functional condition; (3) the phenomenon in question may have a marked significance for prognosis. In a peripheral paralysis of the radial nerve, the presence of the passive index phenomenon, when it has been absent previously, indicates the beginning of improvement; in central organic paresis, the disappearance of the active index phenomenon, when it has previously been present, indicates a very mild degree of paresis.

JONES, Detroit.

PSYCHIC DISTURBANCES AND HYPOPHYSIAL TUMORS. PAUL SAINTON and NOEL PÉRON, Encéphale **18**:358, 1923.

In connection with a rather large epithelioma of the hypophysis without glandular or ocular signs, but with distinct radiographic evidence and mental changes, Sainton and Péron take up the various types of psychoses met with in hypophysial disorders. It has long been recognized that the mental condition of giants is abnormal. Schuster states that 65 per cent. of cases of pituitary tumor are accompanied by psychic disturbances. The case reported is of a man, aged 48, who complained of violent headache, difficulty in walking and failing memory. In general he lay inert, taking no interest in his surroundings, though at times he became jovial. He tired easily, his speech became slower and he reverted to pleasant revery. Intellectual activity and memory were greatly affected, although he remembered music well. Affectivity was practically absent.

The necropsy revealed a tumor measuring 37 x 34 mm. pushing up from the sella, compressing the third ventricle, and, in one place, actually invading the cerebral tissue. In the neighborhood were dilated perivascular spaces filled with round cells.

The authors class the psychoses arising in the course of pituitary tumors as follows: (1) Mental states (acromegaly, gigantism, adiposogenital syndrome); (2) true psychoses; (3) variable psychic disorders in which a state of dementia is common and more or less marked.

In acromegaly there are inaptitude for work, lassitude, modifications of character, depression with asthenia, and a melancholic tendency. Intellectual debility is shown especially by inability to elaborate. In cases of adiposo-

genital dystrophy there is arrest of psychic as well as of physical development. In older subjects apathy and somnolence predominate. True psychoses have the forms of the common mental syndromes. Psychic troubles with a tendency toward dementia are recognized by torpor, imperfect memory and judgment, confusional states and sometimes, as in the case reported here, a mixture of torpor with euphoria.

In dealing with the cause of these troubles the authors consider endocrine, mechanical, and psychoregulatory extracortical centers. The resemblance of the symptoms to those of tumors of the frontal lobe suggests that local pressure may be the cause.

FREEMAN.

THE PHENOMENA OF RAYNAUD'S DISEASE. J. ARTHUR BUCHANAN, Am. J. Med. Sc. **164**:14 (July) 1922.

Buchanan reviews the local manifestations associated with Raynaud's disease and analyses the histories of sixty-seven patients observed in the Mayo Clinic. Birthplace and place of residence were greatly diversified; twenty-one occupations were represented; physical and nervous strain was not a constant factor. There was a slight predominance of females, 59.8 per cent. The mode of onset in the majority of cases was sudden and characterized by a portion of the body turning "white, blue or black"; in other cases there was noticed paresthesia, clumsiness or discomfort. The local syncopal or asphyxial attacks lasted as a rule from fifteen minutes to one hour. The gangrene usually lasted about two months before sloughing of the part occurred. The lesions were bilateral and symmetrical in 43.2 per cent. The blood picture, blood pressure, pulse pressure, and pulse rate were inconstant. Urine examination was negative in all cases.

Interesting incidental findings are recorded. One patient was always well during her pregnancies. In one patient menstruation was suppressed during an attack. One patient had a transient hemiplegia during attacks. One man was sexually impotent during attacks. Several patients observed that if they pricked their fingers during the stage of local syncope no bleeding occurred. One patient suffered from migraine and during the two years in which she gave evidence of Raynaud's phenomena had eighteen convulsions.

Of the measures for relief a warm non-variable climate seemed to be most helpful. Thyroid extract produced no benefit and the eradication of foci of infection was also without results.

VONDERAHE, Philadelphia.

LETHARGIC ENCEPHALITIS AND HERPES FEBRILIS. Z. SZYMANOWSKI and NATHALIE ZYLBERBLAST-ZAND, Brain **46**:49 (Part 1) 1923.

By injecting material from the vesicles of herpes febrilis into the cornea and beneath the dura in a series of rabbits, the authors showed that this disease was capable of being transferred to the central nervous system; also that emulsions from these brains were infective to other animals. The cerebral changes were manifested by a series of nervous symptoms: unilateral positions of the head, stereotyped circus movements of the body, profuse salivation, stiffness of the jaw, convulsions and paresis. Postmortem examination of the brain always revealed pathologic changes identical with those due to epidemic encephalitis. These results were similar to those obtained by Doerr and Levaditi. By injecting another series of rabbits in a similar manner with the nasopharyngeal discharge, the cerebrospinal fluid, or brain emulsions from

patients suffering from epidemic encephalitis, the same clinical and pathologic picture was produced. The authors conclude that the clinical symptoms of infection by epidemic encephalitis (lethargica) are identical with those of herpetic infection. There is a great variation in the incubation period; in some animals clinical symptoms were absent, but the infection was always detected by postmortem examination. Unlike the symptoms observed in man, the authors never observed in animals spasmodic or catatonic symptoms, tremors, myoclonic movements, Parkinsonian findings, sleepiness, or paralysis of the oculomotor nerves; on the other hand labyrinthine symptoms, and convulsions, general or local, were frequently present. Similar results were produced in healthy rabbits by inoculations from those already infected, but the results were slower to develop and more difficult to obtain. Histologic examination of the brains of rabbits infected by either method described, showed changes typical of epidemic encephalitis; in all cases the pia was involved. The circus movements mentioned are explained by involvement of the vestibular nuclei, the authors believing that the virus in animals has an affinity for this nucleus just as the virus of the human form selects the oculomotor nuclei. In all their animals, infected with either virus, inflammatory changes in the liver, consisting of mononuclear infiltration about the bile ducts, were found. The characteristic changes common to animals and man are: infiltration of mononuclear cells, localization in the basal ganglions, and degenerative changes in the cerebellum.

STACK, Philadelphia.

HUNTINGTON'S CHOREA AND MIGRAINE. BENEDEK and GOLDENBERG, Deutsch. Ztschr. f. Nervenheilk. **78**:100 (April) 1923.

The authors report a family in which Huntington's chorea was associated with migraine. Three generations were studied. The patient observed was a woman, aged 36, suffering from Huntington's chorea of four years duration. She had three sons, the eldest died at the age of 6 weeks; the second, aged 16, is healthy; the third, aged 13, has typical migraine attacks. The father of the patient died at 47 from pulmonary tuberculosis; he was markedly alcoholic and developed Huntington's chorea at 43. The paternal grandparents died at the ages of 95 and 96. A maternal grandaunt was feebleminded and a stutterer. The patient has had seven sisters. The first two died from scarlet fever in childhood; the third, a mother of four children is well, but one son, aged 12, has enuresis; the fourth is the mother of three healthy children; the fifth died at 36 from Huntington's chorea that began at the age of 21; the sixth, aged 44, who is married but has no children, developed Huntington's chorea at 38; the seventh, aged 40, who is married, but childless, has suffered from migraine for twenty-eight years. The eighth child in this family is the patient whose case is reported in detail.

A similar case history of Huntington's chorea with migraine was previously reported by Benedek and Csorsz. The authors question a definite hereditary relationship between the two diseases but report this study in order to stimulate further research.

HAMMES, St. Paul.

INFLUENZAL MENINGITIS. T. M. RIVERS, Am. J. Dis. Child. **24**:102, (Aug.) 1922.

The author reviews 197 cases of influenzal meningitis from the literature and reports twenty-three new cases. An analysis of the entire group is made.

The disease is essentially one of infancy, 79 per cent. of the cases occurring in patients under 2 years of age. The clinical picture is not characteristic; it is often indistinguishable from epidemic cerebrospinal meningitis. A leukocytosis with an increase of polymorphonuclear cells is usual. Positive blood cultures may be obtained; in the author's series eight positive blood cultures were obtained in the eighteen cases examined. The spinal fluid is slightly turbid, later becoming purulent; the spinal fluid with stained smears shows gram-negative pleomorphic bacilli. After analyzing data from histories and necropsies the author finds evidence that in the majority of instances influenzal meningitis is a primary infection and that it is probably a carrier-borne disease. A study of influenzal epidemics shows no increase of influenzal meningitis at this time; likewise the seasonal incidence of influenzal meningitis and of epidemic influenza and pneumonia do not coincide. The author also compared meningitic strains of *Bacillus influenzae* with fourteen respiratory strains; in no instance was any one of the respiratory strains identical with meningitic strains serologically even though they had similar cultural characteristics.

VONDERAHE, Philadelphia.

CASE OF EPILEPSY OF TWENTY-TWO YEARS' STANDING DUE TO CALCIFIED ENDOTHELIOMA OR PERITHELIOMA IN THE LEFT LATERAL VENTRICLE, REMOVAL AND RECOVERY. J. LYNN-THOMAS, Brit. J. Surg. **9**:36, 490 (April) 1922.

This is a report of a case of epilepsy in a woman aged 48. At the age of 24 she had a seizure that began in the right ring finger and later involved the right side. Attacks recurred once a year for fifteen years and then increased in frequency (fourteen day intervals). There was no history of an aura or loss of consciousness until the last six seizures. The attacks were followed by right hemiplegia, which lasted one or two hours. On examination there were: weakness of the right side, with wasting of the muscles; motor aphasia; increased tendon reflexes and a Babinski sign on the right side; diminished sensation to heat, cold, touch, and pain on the right side; loss of stereognostic sense in the right hand; enlargement of the veins of the left fundus oculi. A roentgenogram showed a shadow above the left ear. At the operation, a subcortical calcified mass, 1¾ by 1⅝ inches, was removed from a point just anterior to a line parallel to the posterior border of the mastoid and about ¾ inch above the external auditory meatus. The patient suffered no postoperative seizures and showed a gradual improvement in the neurologic signs. Ten years after the operation she was able to do her own housework and suffered only from athetoid movements of the right hand. Pathologically, the calculus was classified as a calcified endothelioma or perithelioma.

POTTER, Akron, O.

SEROUS MENINGITIS AND BRAIN TUMOR. HENRI CLAUDE and HENRI SCHOEFFER, Encéphale **18**:254 (June) 1923.

Localized serous meningitis following infections and intoxications may be difficult to differentiate from tumor of the brain. The authors report a case in which there were definite evidences of intracranial pressure which had gradually increased over a period of three years. At necropsy, the third and lateral ventricles were dilated. There was an ependymitis in the walls of the

ventricles and an ependymal cell hyperplasia in the spinal cord. There was, in addition, a vascular fibroma about the size of a cherry lying between the lateral portion of the pons and the anterior portion of the cerebellum on the left side. It was separated from the cerebellum by pia and attached to the dura by a pedicle.

Such tumors are seldom the cause of increased intracranial pressure. The authors suggest that the tumor was a source of irritation and that ependymitis was a result of this irritation.

The patient had certain pituitary and metabolic symptoms which may have been due to the distention of the third ventricle. The pituitary body itself, while small, was normal on microscopic examination.

HYSLOP, New York.

EXPERIMENTS IN TREATING EPIDEMIC ENCEPHALITIS WITH CASEIN INTRASPINALLY. M. ROCH, Presse méd. **31**:496 (June 2) 1923.

A 10 per cent. solution of casein in ampules was diluted with physiologic salt solution to 0.001 gm. of casein in 1 c.c. of solution; part of the cerebrospinal fluid withdrawn at lumbar puncture was mixed with a measured quantity of this casein solution and re-injected. One-half milligram of casein seemed the optimum dosage; in one case a second injection (1 mg.) was given.

The injection caused a meningitic reaction, clinically and serologically, with an increased permeability of the meninges to nitrates administered by mouth. It was thought that this increase in permeability should theoretically extend to antibodies produced by the organism against the epidemic encephalitis, and similarly to hexamethylenamin, which was introduced by mouth or intravenously as part of the method.

The puncture alone was credited with producing some increase in permeability. On the other hand, a dose of 2 mg. of casein produced too severe a meningitis. The 0.5 mg. dose apparently gave good results in two cases of the short series, and further trials were recommended.

HUDDLESON, New York.

THE SUBDIVISIONS OF THE NEURAL FOLDS IN MAN. G. W. BARTELMEZ, J. Comp. Neurol. **35**:231, 1923.

A two somite human embryo presents five enlargements of the neural folds: a forebrain, a midbrain, and three hindbrain segments. The midbrain is identified by the cranial flexure. Constrictions separate this segment from the forebrain in front and the hindbrain behind. Anatomical characters are cited which mark these segments. In following the development of these segments up to the sixteen somite stage it is pointed out, in particular, that the first hindbrain segment becomes the trigeminal segment and hence associated with the semilunar ganglion and that the middle hindbrain or otic segment becomes identified with the acusticofacialis primordium.

Evidence is presented that differentiation proceeds from the post-otic region forward; that the hindbrain is the most dominant feature of the brain in the early stages; and that at the beginning of the fourth week of pregnancy the forebrain is not relatively larger in man than in other vertebrates.

GRAY, Chicago.

THE CHARACTERISTICS OF THE CEREBROSPINAL FLUID IN POSTDIPHTHERIC PARALYSIS. Joseph C. Regan, Catherine Regan and Brickhouse Wilson, Am. J. Dis. Child. **25**:284 (April) 1923.

The authors note that clinicians, with the exception of French investigators, have paid scant attention to the characteristics of the cerebrospinal fluid in postdiphtheric paralysis. Sixteen patients were studied. The cerebrospinal fluid was clear in all instances and was under normal or slightly increased tension. A Bordet-Wassermann reaction was negative in all instances. The cell count was always within normal limits. A mildly positive globulin reaction occurred in about one third of the patients, indicating in these cases a dissociation between the cytologic and chemical findings. The colloidal gold reaction gave a positive reaction in the syphilitic zone, occasionally extending into the higher dilutions. The colloidal gold reaction was the most constant finding and occurred in all cases except one; in this case there was a slight pharyngeal paralysis with subsequent recovery.

Vonderahe, Philadelphia.

VENOUS ANGIOMA OF THE CEREBRAL CORTEX. W. Drought and C. A. Ballance, Lancet **2**:125 (Jan. 15) 1922.

These authors remark on the rarity of pure venous angioma of the cerebrum and report the case of A. J. A., aged 31. At 27 years, he first noted tingling of the left side of the face accompanied later by transient loss of consciousness, and followed by loss of power in the left arm and leg. The seizures gradually increased in frequency and severity. On examination, the muscles of the left side were weaker than those of the right and the tendon reflexes were increased. No eye ground or sensory changes were demonstrable. Later weakness of the left lower face, ankle clonus (left), and extension of the left great toe (Babinski) appeared. The seizures were preceded by tingling in the left side of the face and tongue. At operation the subarachnoidal veins were found greatly enlarged over the right motor cortex. Marked improvement with no seizures for five months followed the operation.

Potter, Akron, O.

STUDIES ON THE CEREBRO-SPINAL FLUID WITH AN ACETIC ANHYDRIDE-SULPHURIC ACID TEST. Oswald H. Boltz, State Hosp. Quart. **8**: No. 2 (Feb.) 1923.

The acetic anhydrid-sulphuric acid test is a modification of Liebermann's test for cholesterol. This test was first used by the author for the detection of cholesterol in spinal fluid. The spinal fluid from a case of general paralysis when treated with the above reagent gave a positive reaction consisting of a "blue pink or lilac color." The technic of the test is carefully given. Tables accompanying the article show results obtained by testing the spinal fluid of patients suffering with various psychoses.

The author concludes (1) that this test is predominantly positive in cases of neurosyphilis and 100 per cent. positive in general paralysis; (2) it may prove an index of degenerate nerve tissue and the author is not convinced that the test is specific for neurosyphilis; (3) in psychoses of functional type the test is negative.

Ebaugh, Philadelphia.

A CASE OF GENERAL PARALYSIS HAVING AN EVOLUTION OF SEVENTEEN YEARS. Cornil and Robin, Progrès méd. **38**:169 (April) 1923.

The writers know of only one reported case of general paralysis, uncomplicated by tabes, which had a longer clinical course than the one they report —one recorded by Vallon and Laignel-Lavastine. These exceptional cases should be reported in order to convince the skeptical that they actually occur. Their patient was first examined in 1905, at the age of 43. At that time, the notes stated: "Syphilis at 19; apathy; indifference; speech difficulty; inequality of pupils." Subsequently, there were remissions with partial subsidence of the mental symptoms; the serologic confirmation of the diagnosis was obtained at a somewhat later date and included a weakly positive spinal fluid Wassermann reaction, and 70 cells. The benzoin reaction was negative. Death occurred at 60, 17 years after the onset of the psychosis.

The necropsy report gives confirmatory macroscopic findings and describes also lymphoplasmatic infiltrations in the sheaths of Virchow-Robin, particularly in the corpus striatum. Histologic demonstration of the spirochete in the brain substance is not mentioned.

Davis, New York.

NEUROPATHIC MANIFESTATIONS IN INFANTS AND CHILDREN AS A RESULT OF ANAPHYLACTIC REACTION TO FOODS CONTAINED IN THEIR DIETARY. W. Ray Shannon, Am. J. Dis. Child. **25**:89 (July) 1922.

Shannon calls attention to the frequent occurrence of the exudative and neuropathic diatheses in the same individual and disagrees with Czerny who believes that the association is merely accidental. In eight neurotic children, which the author selected for his study, four presented skin eruptions; in all there were positive anaphylactic reactions to foods contained in the dietary. Improvement occurred in all instances when the diet was properly corrected. In one case it was possible to relieve or bring on the nervous symptoms by removing or adding to the patient's diet the food to which she was sensitive.

Vonderahe, Philadelphia.

TROPHIC CHANGES IN SYRINGOMYELIA. F. H. Kooy, M. J. So. Africa **17**:7, 133 (Feb.) 1922.

This is a report of a case of syringomyelia with prominence of the left half of the forehead, exophthalmos, loss of vision in the left eye (except light perception), loss of pupil reaction to light (left), atrophy of the left disk, thickening of the bony floor of the left orbit, enlargement of the left zygoma and maxilla, thickening of the left half of the hard palate, and blocking of the left nasal meatus. There was dissociation of sensibility (pain and temperature) over the left arm, neck and one-half of the face. At necropsy, in addition to the bony changes noted, the sphenoid, maxillary and frontal sinuses were completely replaced by solid bone. Microscopic examination of the spinal cord showed proliferation of the ependymal cells about the central canal in the dorsal and cervical cord and in the medulla with extension of the cavity of the canal into the left posterior horn. In the upper cervical segments the ascending fibers of the fifth cranial nerve were involved in the process. The author believes that the involvement of the fifth nerve is responsible for the unusual trophic lesions in the case recorded.

Potter, Akron, O.

THE CELL STRUCTURE OF THE SUPERIOR OLIVE IN MAN. EDWARD F. MALONE, J. Comp. Neurol. **35**:205, 1923.

From the character and distribution of the Nissl granules within the cells of the superior olive, Malone has shown that the cells of this nucleus structurally resemble neurons of the motor type. The cells of the reticular formation medial to the superior olive show this resemblance even more closely. The inference is made that this nucleus is an efferent correlation center and that the cells do not send axons to the higher auditory centers.

Malone further finds that the cells of the superior olive and those of the preganglionic visceral nuclei are similar in type. Both are essentially premotor cells. It is suggested that in the tegmental portion of the brainstem, at least, typically afferent cells are probably confined to the primary sensory nuclei and that here, at the first synapse, the nervous impulses pass from cells of afferent type to cells of efferent type; in other words at this point the reflex changes from its sensory to its motor element.

GRAY, Chicago.

CHOREA CRUCIATA. LHERMITTE and BOURGUINA, Encéphale **18**:228 (April) 1923.

The authors present a clinical description of a patient with a muscular disorder, hemiplegic in distribution, with manifestations both of so-called chorea and of cerebellar nature. The distinct cerebellar manifestations justified the authors in placing the lesion in the region of the superior cerebellar peduncle. This cerebellar variety of chorea is quite distinct from chorea of striate origin because of the associated cerebellar symptoms.

HYSLOP, New York.

THE INTRODUCTION OF ANTIMENINGOCOCCUS SERUM BY CISTERN PUNCTURE. REPORT OF A CASE OF MENINGOCOCCIC MENINGITIS IN AN INFANT AGED FOUR MONTHS CURED BY THIS METHOD. A. GRAEME MITCHELL and J. J. REILLY, Am. J. Med. Sc. **164**:66 (July) 1922.

Mitchell and Reilly used the cistern puncture described by Ayer in this case of meningococcic meningitis, when, as shown by a depressed fontanel under normal tension, it was evident that there was a persistent subarachnoid block. Antimeningococcic serum was introduced by this route on two alternate days; on the fourth day after administration, the cerebrospinal fluid had become perceptibly clearer and free from meningococci. The patient recovered and six months after discharge from the hospital presented no symptoms except atrophy of the left eye.

VONDERAHE, Philadelphia.

Society Transactions

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HARVEY CUSHING, M.D., *President*

A CONTRIBUTION TO THE NEUROPATHOLOGY OF THE PARKINSONIAN SYNDROME FOLLOWING EPIDEMIC ENCEPHALITIS.

DRS. ISRAEL STRAUSS and JOSEPH H. GLOBUS.

The frequency with which acute epidemic encephalitis terminates in a clinical picture not unlike the parkinsonian syndrome, and the rarity of anatomic observations made in such cases, gives to the case herewith reported an unusual interest.

Clinical History.—The patient, a man aged 37, except for the accidental loss of his right eye, was well up to February, 1920. He then became suddenly ill. There was a moderate rise of temperature, general body pain, drowsiness, alternating with occasional episodes of delirium. He remained in bed twelve weeks, making full recovery.

Six months later, without any febrile reaction, there began a gradual and progressive development of the outspoken parkinsonian syndrome. At first there appeared a tremor of both upper extremities, soon followed by a similar tremor in the lower extremities; with this there developed rigidity, a tendency to propulsion, masklike expression of the face, pill-rolling movements of the fingers, and profuse sweating. There were no localization signs except some narrowing and irregularity of the left pupil. The condition of the patient remained stationary until December, 1921, when a sudden change occurred associated with a rise in temperature, and swelling and inflammatory changes in the joints. Death took place in January, 1922, with bronchopneumonia and myocarditis as the terminating events.

Gross Anatomy.—The findings here are limited to the distinct and marked narrowing of the substantia nigra, which appeared much lighter than normal in color.

Microscopic Anatomy.—The following positive findings are based on an exhaustive search of the entire brain substance:

1. Cerebral cortex: normal in all regions.
2. Subcortex: only occasional lymphocytic infiltration of blood vessels.
3. Striatum: normal.
4. Pallidum:
   (a) No reduction in the number of cells.
   (b) Only slight occasional degenerative changes in the ganglion cells.
   (c) No increase in fat content.
   (d) Marked reduction in iron content.
   (e) Occasional mild lymphocytic infiltration of blood vessels.
5. Thalamus, nucleus ruber and corpus Luysi: normal.
6. Substantia nigra: most striking and definite changes were found, limited to the zona compacta.
   (a) Striking reduction in ganglion cells.

(b) The cells found in the area showed marked degenerative changes and loss of pigment material.

(c) Marked gliosis with rosette formation.

(d) Frequent perivascular infiltration.

(e) Reduction in iron content.

7. Myelin stains of serial sections did not bring out definite changes in fiber structure.

The principal interest in this case lies in the fact that it presents the clinical picture of paralysis agitans, in a patient who died from encephalitis, and in which the principal lesion consisted in a bilateral destruction of the substantia nigra, with a very slight pathologic involvement of the globus pallidus on one side. Trétiakoff was the first to call attention to the degeneration of the substantia nigra in cases of paralysis agitans. Since that time Goldstein has reported a case of postencephalic parkinsonian syndrome in which the lesion also involved the substantia nigra. Foix has reported a similar case.

McKinley has likewise recently described a case of the same nature developing in epidemic encephalitis, in which there was a lesion affecting principally the substantia nigra.

The changes which occur in our case in the globus pallidus can be regarded as minor in degree, and as not necessarily of such a nature as to lead to loss of the function subserved by this structure. Therefore, it is apparent that the syndrome of paralysis agitans can be due to a destructive lesion in the substantia nigra as well as to changes in the pallidum which have been hitherto described as the cause of this clinical picture.

DISCUSSION

Dr. E. Bates Block, Atlanta: Dr. S. S. Schochet and I examined four cases of Parkinson's syndrome, following acute epidemic encephalitis, with the Aberhalden test. We used the globus pallidus and putamen as substrates. All tests were negative, except in one case in which the first test was positive; on repeating, it proved to be negative.

Dr. Frederick Tilney, New York: Frequently, studies of the brain are so desultory that the results are unsatisfactory. When the method of serial section is applied, we may then speak with some degree of certainty of the pathologic lesion and its extent. I think the authors' conservatism is timely, in sounding a note of caution, lest we accept too readily and wholly any one theory as to the position of a lesion producing the syndrome with which we are familiar, typified particularly by parkinsonian characteristics. Certainly there is abundant testimony to show that other lower centers, mesencephalic particularly, may have a great deal to do with the production of these peculiar hypertonic and agitans states.

As long ago as 1895, Brissaud, in a case of isolated tubercle in the substantia nigra, reached the conclusion that this body had much to do with the regulation of muscle tonus, and perhaps also with automatic associated movements. He even went so far as to make the statement that the substantia nigra, or "locus nigra," as he called it, might well enough be the anatomic substratum of paralysis agitans. Drs. Strauss and Globus, in effect, are bringing into line a well studied case with this particular observation.

Dr. William G. Spiller, Philadelphia: This interesting paper might be emphasized in importance by asking in how many cases of encephalitis epidemica without the paralysis agitans syndrome did he find changes in the substantia nigra? Almost invariably the mesencephalon is the area chiefly affected in this disease, and one would suppose, a priori, that the substantia nigra could

hardly escape. In studying a series of cases microscopically, I had one case of encephalitis, in which the mesencephalon was much less affected than the pons and the medulla oblongata.

It is curious that this paralysis agitans syndrome develops in a number of cases at a considerable period after the disease has passed. Drs. Strauss and Globus will possibly explain that by the overgrowth of the neuroglia in the substantia nigra. Perhaps that is the reason why in their case it was six months before the syndrome of paralysis agitans developed.

They referred to the rubrospinal tract. I should like to know from the members of the Society, especially from Dr. Tilney, who has been working on the anatomy of the brain, what authority there is for attributing so much function to the rubrospinal tract in man? Many years ago it was stated that this tract is much less important in the human being than it is in some lower animals.

DR. TILNEY: There is no question about the fact which Dr. Spiller has mentioned. In lower mammals, it is a tract of far greater importance than the pyramidal tract. A shift to the neokinetic system with the further development of the pallium would probably explain the progressive decrease in size of the rubrospinal tract in the primates and especially in man. By this shifting to the neokinetic structures, some functions, of which the older motor system in the striatum formerly had charge, were then delegated to the pyramidal system. I believe that the rubrospinal tract is important, both in relation to the cerebellum and to the striatum, particularly to the pallium, but in its latter capacity decidedly more so in the lower mammals than in man.

DR. STRAUSS, in closing: In the studies of acute epidemic encephalitis which were made by Dr. Globus and myself, we were never particularly interested in the histologic features, so that I am unable to tell Dr. Spiller whether there was involvement of the substantia nigra.

SOME OF THE CHIEF CHARACTERISTICS OF THE SO-CALLED INSTINCTIVE REACTIONS. DR. STEWART PATON.

The present popular interest in certain phases of psychology has directed attention away from some of the old, important, and unsolved problems of the relations of body and mind. It is unfortunate that the variety and intimacy of these relations are not discussed more frequently by this society. The time has come when the organic neurologist and the psychiatrist should be expected to be more explicit than they are at present in stating their reasons for wishing to be members of the same society. If we were to judge intellectual relations by the small number of papers presented that discuss the relations of body and mind, an outsider might have occasion to affirm that the mutual interests of psychiatrists and neurologists were largely of a sentimental character.

No doubt the present demands made on the psychiatrist for assistance in helping to organize society on a more rational basis are so urgent that there is danger he may not take sufficient time to think about the foundations of the house in which he proposes to live. What are then some of the more important connecting links between the objective minded neurologist, who still owns and uses a microscope, and the psychiatrist completely absorbed in the new dynamic psychology and cultivating only a subjective mental attitude toward the problems in which both investigators should have a common mutual and sympathetic interest.

If we try to describe some of the chief characteristics of the so-called instinctive reactions, we shall find excellent reasons for encouraging more sympathetic

relations between students of structure and of function. The necessity for close cooperation between subjective and objective minded investigators is strikingly, for example, emphasized if we trace the genesis of some of the reactions occurring during embryonic life. These reactions represent stages in the integration of adjustments which later come to be recognized as instinctive in character. Various phases in the integration of these reactions can be correlated with definite structural changes. We seem to be justified, therefore, in assuming that the structural organization to a large extent determines the character of the functional response.

The process of integration of reactions taking place prior to birth, including components that form such important factors in all instinctive adjustments, deserves to be more carefully studied. Doubtless, if the number of factors involved in these particular forms of organized response had been more closely observed we should be more impressed than we are with the arbitrary limitations often imposed on the use of the terms "instinctive reactions." The acceptation of the usual limitations accepted in describing these responses gives a wrong idea about the number and variety of processes involved in instinctive adjustments.

There is no good reason for assuming, as the psychologist often does, that the character of the instinctive reactions is specifically different from processes taking place during embryonic life. For example, it is difficult, if not impossible, to pick out specific differences distinguishing the first bodily movements of the vertebrate embryo occurring at a very early period in embryonic development from the adjustments taking place after birth, at the time when the psychologist claims that the reactions are characteristics of instinct.

When the embryo chick (14 mm.) or guinea-pig (12 mm.) is pricked with a needle, this stimulus is followed by movements of the body away from the stimulus—it results in a protective adjustment. No one would seriously question our right to designate this reaction as a reflex. A little later, however, in the life of the embryo a prick of the needle is followed by a series of what are relatively much more complicated movements, marking the development of the machinery required to speed up as well as to continue movements. In such reactions there are suggestions of the fact that already the embryo is beginning to pay attention to certain forms of stimuli, to remain indifferent to others, and to react in a complicated manner to annoying or painful situations.

If we follow closely these reactions of the embryo as the integrations become more complex we shall find it exceedingly difficult to select any special feature, sufficient in itself to differentiate the so-called automatic adjustments occurring before birth from the reactions of the new-born animal when, for example, it is said to seek nourishment instinctively.

It seems therefore to be merely a matter of personal preference whether the integration of responses has reached a degree of complexity when they shall be described as automatic movements, or shall be regarded as forms of adjustment usually described as instinctive responses.

Long before birth, the thyroid and suprarenals, part of the machinery necessary for strengthening and continuing simple movements, if the exigencies of the situation demand greater and more complicated efforts, are well supplied with nerves. These organs seem to be ready to take an active part in increasing the capacity of the organism to adjust its life to more varied and complex situations.

The concentration of effort exclusively on the attempt to give a purely psychologic account of instinct either in man or in any of the other vertebrates,

without considering the structural organization is liable to make us overlook important events occurring in connection with the earliest reactions of the living organism.

If we study the living embryo carefully, it is easy to understand how very slight anomalies of structure, such as variations in the development of the thyroid and suprarenals, may be responsible for considerable variations in the instinctive reactions. Even very slight structural changes can be responsible for the development of muscular attitudes that are of great importance when considered in connection with the development of the entire emotional life. Suppose, for example, that there exists at birth even a slight degree of hyper-excitability of the thyroid and suprarenal functions. One result of these conditions would be that the very young animal would at once develop a motor attitude toward the extra-uterine environment, that on very slight provocation will express the beginning stages of apprehensiveness, fear, or fright. The young animal is predisposed by its physical organization to react emotionally in certain definite ways that in the case of the human subject may affect the development of the entire mental life.

A close study of the structural organization of embryos clearly indicates that the predisposition of the young animal or infant to develop early in life states of fear and apprehensiveness can often be accounted for on a physiologic basis. In many instances the psychologic factor is only of secondary importance. Some animals and people are literally born to show symptoms of fear on every possible occasion.

We should not forget, however, that one set of habits formed prior to birth may be inhibited or interfered with by quite another set induced by the new extra-uterine environment. The cutting out of one set by a second set of habits is a process essentially different from what would occur if a hypothetical censorship or repression of impulses were the deciding factors in determining behavior.

The impulses of the growing organism are discharged through a variety of channels. We can easily imagine them to be switched quickly from one line of discharge to another. The operation involved in switching is quite different from that which is supposed to take place in the act of repression.

It has seemed to the writer to be desirable in studying "frustrated impuses" to remember that, as far as is known, the shunting or side-tracking of one impulse by another, not repression, is what is observed in connection with nervous impulses.

Doubtless, as we become more fully acquainted with the organization of the motor activities of the embryo we shall be in a better position, not only to understand the instinctive reactions, but also to pick up interesting clues as to the nature of attitudes and tendencies expressed by wishes, and even to clear away some of the mystery now associated with the unconscious.

We have become so accustomed to studying only the particular phases of the phenomena in which we happen to be interested that we seldom stop to consider their relation to other biologic processes. This objection is particularly noticeable in connection with recorded observations on the instinctive reactions. Freud's recent definition of an instinct as "an innate tendency in living organic matter impelling it toward the reinstatement of an earlier condition" represents only one phase of a large problem. Braun has done well to call attention to the fact that the instinctive responses of the organism are not only the continuation of, but are identical with organic development. Once we grasp the full biologic significance of this idea, we are in a better position to begin to investigate the nature of the forces shaping the personality.

DISCUSSION

DR. SMITH ELY JELLIFFE, New York: In trying to think out some of the antenatal influences that might modify conduct, and in the course of a discussion before a medical society apropos of musical talent and its relation to rhythmic stimuli and their possible conditioning by the mother's heart-beat during the fetal period, I threw out the inquiry that I should like to know what was the nature of Irving Berlin's mother's heart-beat. Could a master of "jazz" music have been "conditioned," i. e., so far as rhythm was concerned, by an irregular heart-beat during fetal life?

Book Review

DISEASES OF THE NERVOUS SYSTEM; A TEXT BOOK OF NEUROLOGY AND PSYCHIATRY. By SMITH ELY JELLIFFE, M.D., PH.D. Formerly Professor of Psychiatry, Fordham University, New York, and formerly Adjunct Professor of Diseases of the Mind and Nervous System, New York Post-Graduate School and Hospital, and WILLIAM A. WHITE, M.D., Superintendent of St. Elizabeth's Hospital, Washington, D.C.; Professor of Nervous and Mental Diseases, Georgetown University; Professor of Nervous and Mental Diseases, George Washington University, and Lecturer on Psychiatry, U. S. Army and U. S. Navy Medical Schools. Cloth. Fourth edition. Price, $9.50. Pp. 1,119 with 475 engravings and 13 plates. Philadelphia: Lea and Febiger, 1923.

Whatever attitude may be adopted toward the interpretations that have been placed on the results of psychanalysis and the formulations that have been built on them, there will be no hesitation in conceding that an intelligent grasp of the consequences of nervous disease is possible only when the functions of this system as an integrating mechanism are fully recognized. The human organism is a living unit, motivated by forces and working with mechanisms many of which are infinitely more primitive than consciousness and must be given consideration in studying the reactions of the man.

In this book the authors have endeavored consistently to pivot their descriptions of the diseases of the nervous system around this conception of the organism as a reacting unit, and to regard the syndromes that develop as expressions of some disorder in integration. This view gives the book a characteristic stamp and makes of it something more than an ordinary text book. The material has been divided into sections corresponding with stages of evolution in the reactive mechanisms. The most primitive are the physicochemical mechanisms which comprise the vegetative and endocrinal integrations, and with them are included the primary disorders of striated muscle. An immense mass of detail is given concerning the anatomy and physiology of these structures in much condensed form which is liable to be confusing to the student, even though it is admirably illustrated with diagrams and photographs drawn from many sources. The relation of these primitive reactive mechanisms to the dynamics (including conscious feelings and the psychanalytic "unconscious") of the organism is strongly emphasized and the foundation is laid for its use in interpreting functional disorders and diseases of higher levels, as well as to explain the possible origin of many somatic diseases. In the main the statements are conservative, though they touch on topics that are admittedly controversial and even speculative. Under this heading are described many syndromes arising from damage to special glands and particular vegetative nervous regions.

The second level is that of the sensorimotor system in which the subdivisions are necessarily conventional. The illustrations are excellent and the material has been brought thoroughly up to date. Here again the effort to be concise and yet to include all recent work often tends to make the language difficult to follow. This fact is evidently recognized by the authors and frequent references are given to original contributions and reviews with advice for further reading. The final level is the psychic or symbolic and includes descriptions

of the psychoneuroses, psychoses and the various forms of mental deficiency, both intellectual and temperamental. In discussing the dynamics of the functional disorders, the formulation is essentially that of Freud. The mechanisms are briefly and simply described, but the strivings, the conflicts and the evolutional levels are all expressed in terms of erotic libido. In a text book of this kind it would certainly seem desirable that the existence of other methods of formulation should be mentioned; the dogmatic assertions detract considerably from the unquestionable merits of the book. The introductory chapters on the methods of examination, both bodily and mental, would prove of particular value to the undergraduate student; but the lack of clearness and the wholesale inclusion of much that is not established render its general adoption by such students without careful guidance, inadvisable. The more advanced student who has acquired the foundation on which to select and think for himself will find this book a useful supplement to standard works.